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**SCIENTIFIC CRITERIA DOCUMENT
FOR
MULTIMEDIA ENVIRONMENTAL
STANDARDS DEVELOPMENT - LEAD**

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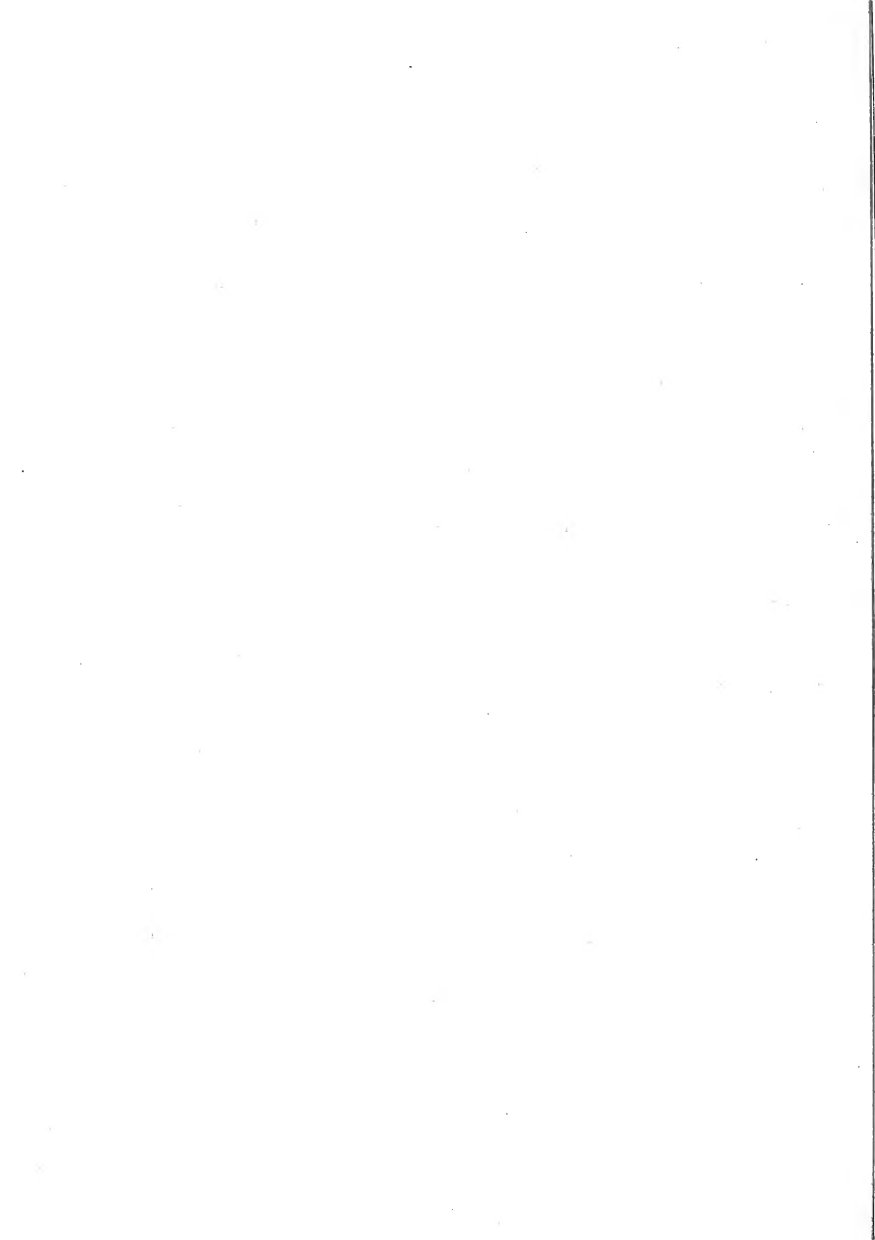


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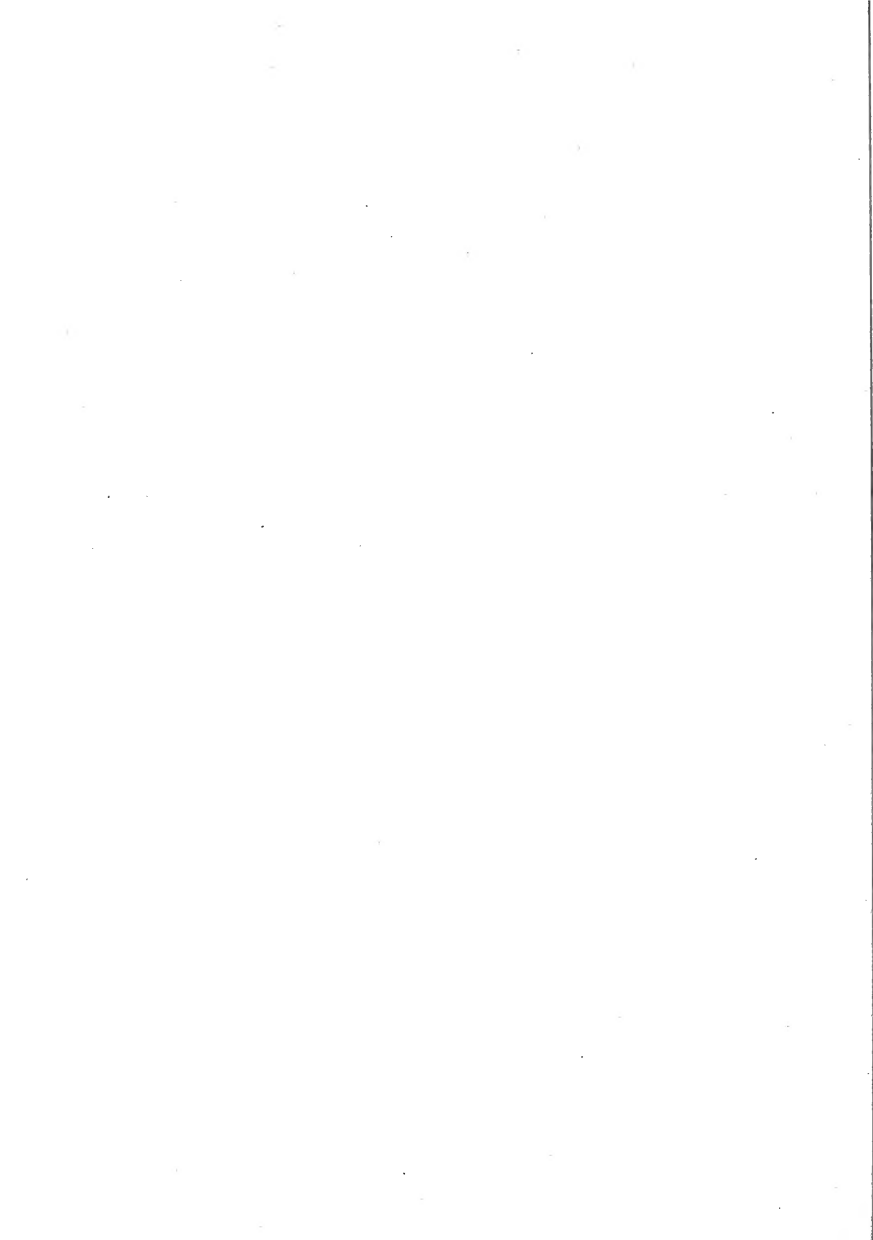
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LIST OF ABBREVIATIONS

ADI	Acceptable Daily Intake
ALA	Aminolevulinic Acid
ALAD	δ -Aminolevulinic Acid Dehydratase
ALAS	δ -Aminolevulinic Acid Synthetase
ATSDR	Agency for Toxic Substances and Disease Registry
BAS	British Ability Scales
BG	Bender Gestalt
BNAS	Brazelton Neonatal Behavioural Assessment Scale
CDC	Centers for Disease Control
CEC	Commission of the European Communities
CNS	Central Nervous System
ECAO	Environmental Criteria and Assessment Office
EC ₅₀	Effective concentration in 50% of the exposed population
EP	Erythrocyte Protoporphyrin
FEP	Free Erythrocytic Porphyrin
GFT-I	Version of Bender Gestalt
GFT-N	Version of Bender Gestalt
G-R	Graham-Rosenblith Behavioural Examination for Newborns
GSD	Geometric Standard Deviation
HOME	Home Environmental Quality Measure
IARC	International Agency for Research in Cancer

IgA	Immunoglobulin A
IgG	Immunoglobulin G
IOC	Intake of Concern
IQ	Intelligence Quotient
IU/BK	Integrated Uptake Biokinetic Model
LC ₅₀	Lethal concentration to 50% of the exposed population
LOAEL	Lowest Observed Adverse Effects Level
LOEC	Lowest Observed Effect Concentration
LOEL	Lowest Observed Effects Level
MATC	Maximum Allowable Toxicant Concentration
MDI	Mental Development Index
MEP	Multimedia Exposure Profile
MFO	Mixed Function Oxidase
MOEE	Ontario Ministry of Environment and Energy
MSCA	McCarthy Scale of Children's Ability
NAAQS	National Ambient Air Quality Standard
NBAS	Brazelton Neonatal Behaviourial Assessment Scale
NCS	Nutrition Canada Survey
NCV	Nerve Conduction Velocity
NHANES	National Health and Nutrition Examination Survey
NOAEL	No Observed Adverse Effect Level
NOEC	No Observed Effect Concentration

OAQPS	Office of Air Quality Planning and Standards
OMAF	Ontario Ministry of Agriculture and Food
OMOH	Ontario Ministry of Health
OSWER	Office of Solid Waste and Emergency Response
PbB	Blood Lead
PDI	Provisional Daily Intake
PKC	Protein Kinase C
PTWI	Provisional Tolerable Weekly Intake
PWQO	Provincial Water Quality Objective
RfD	Reference Dose
S-B IQ	Stanford-Binet Scale of Intelligence
SEGH	Society for Environmental Geochemistry and Health Model
SES	Socio-economic Strata
SMR	Standard Mortality Ratio
TM	Trail-Making Test
USEPA	United States Environmental Protection Agency
USFDA	United States Food and Drug Administration
VRD	Vienna Reaction Device
WHO	World Health Organization
WHO/EURO	World Health Organization, Regional Office Europe
WISC	Weschler Intelligence Scale
ZPP	Zinc Protoporphyrin

EXECUTIVE SUMMARY

Lead holds a unique position among the known environmental poisons. Knowledge of its threat reappears throughout history from ancient times to the present. Extensive industrial and domestic use over several centuries has led to its release and distribution throughout the environment. Lead is also a persistent multimedia contaminant. It is present in diverse environmental media and exposure may occur from several sources and pathways. Lead is therefore regulated in many different pathways, including ambient air, drinking water, soil, sludges, sediment and consumer products. The importance of lead as a contaminant of concern is reflected in the extraordinary volume of scientific literature devoted to its toxic effects as well as the large degree of regulatory efforts by this and other agencies. This scientific criteria document is an attempt to chronicle the current knowledge on the human health effects of low level lead exposure and to provide a detailed risk assessment of environmental exposures in Ontario. This information is designed to be applied to the development of a protective set of standards for air, drinking water and soils, as well as any other appropriate regulatory initiatives concerning lead.

This project was undertaken for two main reasons. First, effective standards and guidelines for lead required a multimedia assessment specific to Ontario. Secondly, there is a recognized need to capture the rapidly evolving knowledge of low-level effects of lead in sensitive populations and reevaluate standards accordingly. Many existing standards, even those developed in the 1980's, require review in light of the information collected in the last few years. As the blood lead levels of concern continue to be lowered, prevention measures such as standards (i.e. prevention before a child is poisoned) must receive more emphasis. Although a number of centralized regulatory efforts in Canada and the United States have had notable effects in reducing lead exposure, the latest scientific data suggest concern at exposure levels previously regarded as safe. The very high priority of lead as a contaminant of environmental concern was recently reaffirmed by the Department of Health and Human Services of the Agency for Toxic Substances and Disease Registry (ATSDR). Based on risk considerations of toxicity and the potential for population exposure, lead was ranked first among priority hazardous substances for assessment.

Hazard identification is the qualitative identification of the potentially adverse impacts of lead, particularly to human health. Critical endpoints and potentially sensitive populations are thus identified, based on information gathered from primary literature, comprehensive critical reviews, agency reports and computer databases.

Lead accumulates in human tissues and affects multiple organ systems. Key targets are the central nervous, hematopoietic, reproductive, renal and cardiovascular systems. Because the central nervous system is especially vulnerable during early development, the fetus and children under the age of six years are the most sensitive populations. Young children are at greater risk of lead exposure for several reasons: they absorb lead more readily than adults; they have more hand to mouth activity; and their intake of lead on a body weight basis is

greater. Placental transfer of lead occurs in humans as early as the twelfth week of gestation, and uptake of lead by the fetus continues throughout development.

Several recent, well-designed epidemiological studies provide evidence that elevated lead exposure during the prenatal and postnatal period is associated with neurobehavioural changes, decreased cognitive abilities, IQ (Intelligence Quotient) deficits and other developmental effects. The causal relationship has been the subject of considerable scientific debate. However, the evolving pattern and consistent findings of independent positive studies give strong support for causation. It is not yet known whether these lead-associated effects are irreversible, but a recent study suggests the effects may persist into young adulthood. Currently no threshold has been defined for the neurobehavioural and developmental effects of lead. Considerable scientific evidence indicates that adverse effects may occur at blood lead levels as low as 10 µg/dL. These impacts are best represented by a shift in the distribution of IQ or MDI (Mental Development Index) scores within the population, rather than as identifiable adverse effects in individual children.

Based on animal tumour data, lead is a probable human carcinogen, which exhibits some "genotoxic" characteristics although genotoxicity cannot be established. Several independent studies have reported a high incidence of kidney tumours in rodents of both sexes when the animals were exposed to elevated doses of lead. The renal carcinomas were distinct from those produced by other substances, but they were observed at doses much higher than would be normally encountered in the environment. The mechanism of kidney tumour induction is not well understood although there is some evidence that lead exhibits genotoxic characteristics. The general absence of tumours at low levels of exposure remains to be explained.

Neurotoxicity and neurobehavioural effects of lead in young children and infants are identified as the most sensitive endpoint of lead toxicity in the most sensitive populations. Fetal exposure, and pregnant women (as surrogates of fetal exposure), are also groups at higher risk. These potential risks are based on studies of children environmentally exposed to lead, not on theoretical extrapolations from laboratory studies or high-dose occupational exposures. Quantitative risk estimates of lead's carcinogenicity could be made, based on animal data and estimates of cancer potency. However, the sizeable uncertainty associated with such extrapolations and the extensive human database available for other health effects of lead, argue against employing such extrapolations for establishing environmental regulations.

Short-term exposures may result in deposition of lead in brain and bone and subsequent exposure to lead released from these tissues may occur in the absence of environmental exposure. Also the neurobehavioural consequences of lead exposure may be linked to sensitive sequential developmental periods. There is no duration of exposure to lead that has been identified as without effect. There is evolving evidence, both human and animal, which is consistent with a small effect on blood pressure, particularly in middle-aged men. The human epidemiological observations present both positive and negative findings, and as such

it is difficult to draw firm conclusions, particularly as they relate to the dose-response function at very low blood lead levels. No threshold for this effect is indicated by current studies. It is believed that characterization of risk for the purposes of standards development is most relevantly focused on developmental impacts in children and such derived guidelines would likely minimize this type of potential effect in adult groups.

Dose-response assessment is the examination of the quantitative relationship between the magnitude of exposure from different lead exposure routes and the occurrence of health effects. The focus of this component is upon the relationship between dose (as blood lead level or intake) and various health responses.

Taken together current studies indicate a general, quantitative relationship between blood lead levels in children and various adverse outcomes. A discrete dose-response relationship cannot be precisely established, particularly for the most subtle effects. There does however appear to be a convergence of initial effects at blood lead levels in the range of 10 to 15 $\mu\text{g}/\text{dL}$ and possibly at lower levels. However, the available data do not allow identification of a clear threshold. Blood lead levels as low as 10 $\mu\text{g}/\text{dL}$ in infants, children and the fetus are associated with adverse neurobehavioural and cognitive changes. This level is therefore identified as the Lowest Observed Adverse Effect Level (LOAEL) for sensitive populations. In adults, effects on blood pressure have been observed across the range of blood lead levels studied, again with no evidence of a threshold.

The lack of a discernible threshold for neurobehavioural effects in children precludes the development of a traditional Acceptable Daily Intake (ADI) or Reference Dose (RfD) intake. Neither of these type of toxicological benchmarks may be regarded as entirely safe or acceptable in the case of lead, because even minute exposures may be associated with a some degree of risk. However, the blood lead level of concern (10 $\mu\text{g}/\text{dL}$) in young children, which represents a "threshold" for U.S. and proposed Canadian intervention strategies may be related to total intake to identify an intake of concern for individuals (IOC_{ind}). On a population, rather than an individual basis, an intake level of concern (IOC_{pop}) is derived based upon the LOAEL of 10 $\mu\text{g}/\text{dL}$, quantitative oral intake - blood lead relationships and application of an uncertainty factor. The recommended intake of concern to be used in the derivation of multimedia guidelines that will be protective of sensitive populations is 1.85 $\mu\text{g}/\text{kg}/\text{day}$ or 0.0018 $\text{mg}/\text{kg}/\text{day}$. This is equivalent to a daily intake of about 24 $\mu\text{g}/\text{day}$ in a child aged 0.5 to four years. The IOC should not be regarded as an acceptable intake or as a safe level below which no effects will occur, because a threshold for critical effects has not been scientifically established. It should be regarded as a level which, if applied to the general population, should offer some measure of protection to individual children. It is likely that the majority of children would have blood leads below intervention levels at this intake limit. This also provides an appropriate measure against which to compare average or typical exposure estimates, which are themselves a measure of central tendency. Exceedance of this intake may not necessarily precipitate adverse effects in an individual child, but may be viewed as a mean intake not to be exceeded in the community.

In utero exposures may occur at highly sensitive periods in organ or organ system development. Estimating the lead intake to blood lead level relationship for pregnant women is complicated by the possible mobilization of lead from past exposures; in other words, lead which has accumulated in internal storage pools such as bone. Fetal exposures are probably dominated by maternal blood lead stores from past, higher level exposures. Therefore an equivalent derivation based on maternal exposures is not supportable. However, because adults generally have lower uptake coefficients for lead than children, intake at the derived IOC_{pop} is not likely to contribute adversely to body burden in pregnant women. The value derived here is somewhat more conservative than ADI values developed by other regulatory jurisdictions.

The dose-response information on either IQ measures or developmental indices do not provide clear dose-response relationships. With respect to developmental effects, prenatal exposure may be of more significance than postnatal exposure. In studies reporting prenatal effects the slope appears to be roughly -0.5 points per $\mu\text{g}/\text{dL}$ and in those reporting postnatal effects, it appears to be -0.3 points per $\mu\text{g}/\text{dL}$. These slopes must be viewed as tentative but could perhaps be used as a crude measure of the effectiveness of various target blood lead levels in scoping the magnitude of impacts being considered.

Exposure assessment requires the estimation of the routes and extent of exposure to lead from all environmental sources by means of modelling or measurement of lead in all environmental media. The multimedia approach which considers total exposures from all environmental media was used to evaluate human exposure to lead. This approach also includes discussion of the results of important epidemiological surveys of blood lead levels in Ontario child populations. Emerging exposure modelling approaches in the scientific community are also reviewed.

Based on epidemiological surveys and an analysis of integrated pathway exposures, a considerable reduction in general lead exposure has taken place in Ontario since the mid-1980's. In 1990, blood lead levels in Toronto children, removed from an industrial point source, averaged approximately $4 \mu\text{g}/\text{dL}$. A significantly higher mean of $12.0 \mu\text{g}/\text{dL}$ was found for urban children in the 1984 Ontario Blood Lead Study. Estimated intakes of lead for young children, based on a multimedia model, have declined by at least 50% over the same period. The declines may be attributed to the phasing-out of lead in gasoline and voluntary elimination of lead solder in food canning processes.

Daily lead intakes from all major pathways for urban children are estimated to be $37 \mu\text{g}/\text{day}$ or $2.87 \mu\text{g}/\text{kg}/\text{day}$. The corresponding adult intake (which includes pregnant women) is estimated at $53 \mu\text{g}/\text{day}$ or $0.75 \mu\text{g}/\text{kg}/\text{day}$. The exposure profile for children indicates that intakes from food represent about 48% of exposure ($17.7 \mu\text{g}/\text{day}$ or $1.4 \mu\text{g}/\text{kg}/\text{day}$). Based on trend analysis, however, this may be an overestimate. Exposure from soil and dust was quantitatively assessed using observed and projected trends in soil and dust levels based on recent reductions in ambient air lead levels. Although use of leaded gasoline has declined markedly, previous use has resulted in widespread contamination of soil and dust. An

average daily intake of 16 µg/day or 1.23 µg/kg/day is estimated for children or about 43% of total intake. Drinking water represents about an average of 8% of total intake (3 µg/day or 0.23 µg/kg/day). Finally, direct inhalation of lead, is a relatively minor direct exposure route estimated at 0.13 µg/day or 0.01 µg/kg/day. Dermal absorption of lead is negligible. This information is utilized to develop a Multimedia Exposure Profile (MEP) for young children and adults.

Estimated daily intakes of lead from food, based upon the summation of 112 food grouping intakes were developed. These dietary lead intakes are based upon dietary data collected in Canada in 1985. Based on the observed significant declines in blood lead which have been observed, the post 1990 phaseout of lead in gasoline, voluntary changes in the food processing industry and observed trends in the U.S. projected dietary intakes for 1993 are developed. This estimate is limited as it is not based on actual analysis of current food baskets. Current total estimated daily intake estimates for 1993 children may be 1.92 µg/kg/day and 0.35 µg/kg/day for adults.

There are various scenarios which may result in higher exposures for certain subpopulations. These may be either chronic or intermittent acute exposures which occur against the background of typical exposure. For example, urban children living in the vicinity of industrial point sources of lead emissions or children living in areas of higher plumbosolvency are expected to be more highly exposed. Estimates of total daily exposure under these scenarios are provided.

Exposure from consumption of home grown vegetables is assessed at various soil levels and it is concluded that this indirect route can contribute lead intakes as large as direct ingestion of soil. Therefore consideration of this exposure in developing soil-related guidelines would appear essential.

For any child, exposure to lead through certain consumer products such as lead-based paints, ceramic glazes, or hobbyist materials can be potentially hazardous with a risk of clinical poisoning. Modelled exposure scenarios indicate that such intermittent exposures may produce exposures much greater than all other environmental exposures combined. Renovation of homes containing lead-based paints can create conditions of high exposure for children and women of child bearing age in Ontario. Weathering or flaking of exterior lead-based paints may also lead to unsafe levels of exposure. These exposures are therefore of concern for Ontario children.

Risk characterization is the integration of exposure and dose-response assessment to determine the nature and magnitude of the risk of exposure to lead. It involves integrating the exposure estimates for various population groups with dose-response information; identifying those groups at greatest risk; and estimating the number of Ontario children potentially at risk.

The groups at greatest risk from the effects of low level lead exposure are young children, the fetus, pregnant women and women of child-bearing age (as surrogates for fetal exposure). Various segments of these groups may experience higher risk as a result of increased exposures through living in the vicinity of point sources, areas of highly plumbosolvent drinking water, or homes containing a lead paint hazard or through individual behaviours like consuming backyard vegetables, or high levels of soil ingestion in particular children. Limited evidence suggests that native peoples may constitute a special population group at risk of higher exposure to lead.

Before discussing the quantitative aspects of risk, some consideration should be given to the severity of the risks under examination. The health significance of a 4 or 8 point reduction in IQ or MDI score may at first appear to be relatively minor. The standard deviation of the normal distribution of scores on the Bayley MDI index is approximately 16 points. Therefore, it could be argued that such an effect is not significant for an individual as it lies within the normal variability of the test method or the effect is so subtle to be considered truly adverse. However, these seemingly small effects may be of considerable significance on a population basis. Statistically, a decline of 5 points in mean IQ score, assuming that the distribution is maintained, results in a greater than two-fold increase in the percentage of children with an IQ below 70 (the level at which a child is deemed to be mentally retarded) and a decrease in the number of children in gifted categories. It has been predicted that a shift of 6 points in the median score is associated with a four-fold increase in the risk of IQ below 80. This shift truncates the upper end of the curve with a 25 % reduction of children with IQ greater than 125 (superior function).

Another important and consistent observation in the studies is that the association of social indicators, such as social class and home environment, are always stronger than any lead/IQ association. The extent to which these characteristics confound the results is not certain. However, a factor which may influence the overall risks for Ontario children is that of changing socioeconomic circumstances. Studies of normal populations in several different studies have confirmed the relationship of lead measures with social indicators of disadvantage. Lack of suitable play areas, missed meals, and low standards of family hygiene are all factors which may contribute to increased lead burden. In 1990, roughly one in six Ontario children lived in poverty. The living circumstances of these children may place them at increased risk of lead exposure.

In characterizing the risks, based on the present exposure assessment for lead for Ontario children, the following conclusions should be considered in regulatory application:

- Exposure of children to lead has declined in Ontario by at least 50% since the early 1980's, as evidenced by comparing the results of limited blood lead survey work carried out in Toronto in 1990 to those of the 1984 and 1987 Ontario blood lead surveys.

From the modelled exposures, typical young children (0.5-4 years) are estimated to have total multimedia intakes of roughly 1.5 times the derived intake of concern for this population of 1.85 µg/kg/day. However the level of dietary intake (and thus total exposure) may have significantly declined in recent years as evidenced by blood lead studies. If the projected value based on American dietary exposure figures is assumed, total daily exposures would very slightly above the suggested intake of concern. Breast fed neonates are estimated to have total intakes below the IOC_{pop} whereas those fed canned or cow's milk may have total intake at or above the IOC_{pop} .

The exposure estimates strongly suggest that, for typical urban children, the margin of safety between actual exposure and intakes associated with concern for potential neurobehavioural effects is small, if it exists at all. Under different higher exposure scenarios, total daily intakes may well exceed the intakes of concern. Major reductions have been realized in lead exposure, but at the same time the blood lead level of concern and the tolerable daily intakes of lead have been reduced by a roughly equivalent margin. Therefore, while actual levels of risk have declined substantially and may decline more, the degree of risk has not changed substantially. Rather, the previous risk level was underestimated by at least a factor of two. Further blood lead surveys of Ontario children will continue to provide the best picture of lead exposure in the 1990's.

Overall, there are moderately small areas of uncertainty in both the dose-response and exposure assessment areas as outlined. The overall confidence in this assessment comparable to most other contaminants is high.

How many children may be currently at risk to elevated exposures? The projected number of Ontario children aged one to 4 years is 564,900. It is assumed that 80% (500,000) live in urban locations. If the recent Toronto surveys are indicative of the current blood lead distribution in urban areas, then about 4% of children (18,000) are estimated to have blood lead levels greater than 10 µg/dL, the blood lead level of concern. This number is greater if children age five and six are included in the estimate.

It is difficult to estimate the extent of the lead in paint hazard or to quantify the numbers of young children potentially exposed in Ontario. If one assumes that private dwellings built prior to 1970 may contain lead paints, this would represent 2,056,850 private dwellings in Ontario.

There are a number of observations stemming from this assessment which may bear on the *risk management* activities of multimedia allocation and the setting of the standards:

For the appropriate and effective development of a set of health-based multimedia guidelines and strategies for exposure prevention and reduction, the Ministry should ensure that all program areas utilize a single consistent scientific approach and toxicological assessment. Standards development is an essential component of an

overall strategy, but other companion approaches are required. The latter may not necessarily fall into the regulatory purview of the Ministry. However, the multimedia assessment can provide useful information which may influence decisions in these related areas.

- The most appropriate focus for regulatory action is prevention of potential neurobehavioural effects in young children and assessment of risk should be based on this. Measures protective of children would generally ensure that environmental lead intakes and resultant blood lead levels remain low enough to limit risk in the vast majority of the remaining population.
- The goal of all lead exposure prevention should be to reduce children's blood lead levels below 10 µg/dL, the recently recognized blood lead level of concern.
- The development of specific health-based guidelines should utilize the derived intake of concern for child populations (IOC_{pop}) of 1.85 µg/kg/day. This figure incorporates the population-based significance of the health effects and attempts to minimize the predicted number of children with individual blood lead levels of concern.
- The Multimedia Exposure Profile (MEP) for young children, as opposed to other age groups, is the most appropriate framework for decisions regarding the allocation of exposure to specific pathways. All related lead guidelines should be considered together, given that the risk assessment indicates an extremely small margin of safety and incremental reductions of lead in single media take on added significance.
- The ultimate *allocation* of the IOC_{pop} to exposure pathways involves decisions beyond considerations in this assessment. In setting guidelines for lead, the other factors, such as analytical detection limits, technical feasibility of control, and economics will be considered. Particular values therefore are not recommended in this document. However, as a guiding framework, and starting point for these decisions, the exposure profile for children is used to derive media-specific doses (Table E.1 and E.2).

Consideration of the available exposure information provides various options including the following:

Option 1: If the relative contributions are applied directly to the IOC_{pop} , this would translate into media-specific doses in E.1.

TABLE E.1 MEDIA-SPECIFIC DOSES BASED ON IOC_{pop}

MEDIA OR SUBSTRATE	RELATIVE CONTRIBUTION (%)	CORRESPONDING INTAKE ($\mu\text{g}/\text{kg}/\text{day}$)
Food	48	0.88
Drinking water	8	0.15
Air	<1	0.02
Soil/dust	43	0.79

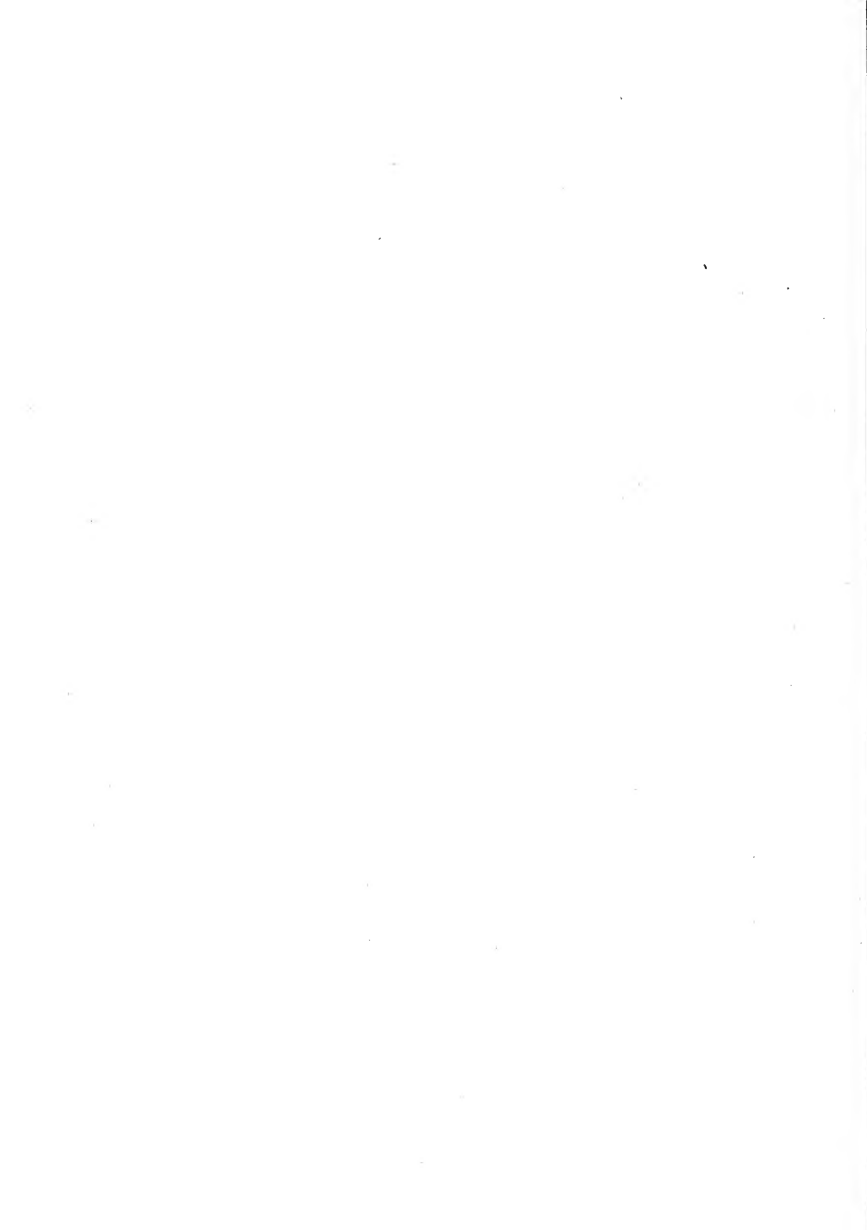
Option 2: If the food intake estimates are adjusted downward on the basis of projection to 1992 as opposed to using actual 1985 food data, this would translate into the media-specific doses for a typical urban child (Table E.2).

TABLE E.2 MEDIA-SPECIFIC DOSES USING PROJECTED 1993 FOOD DATA

MEDIA OR SUBSTRATE	RELATIVE CONTRIBUTION (%)	CORRESPONDING INTAKE ($\mu\text{g}/\text{kg}/\text{day}$)
Food	24	0.44
Drinking water	12	0.20
Air	<1	0.01
Soil/dust	64	1.18

Other approaches could also consider allocation at potential sources (e.g. the input of air lead to soil lead) as opposed to allocation at the receptor.

The lack of a discernible threshold and associated reference dose for lead presents a unique situation with respect to the application of quantitative risk assessment approaches to develop health-based criteria for lead. However, the quantity and quality of toxicity information is such that it would be difficult to argue that a risk assessment approach to lead criteria should not be followed to some degree. As with non-threshold carcinogens, it must be recognized that any measure or guideline may still be associated with some degree of risk. The IOC_{pop} and doses suggested here are presented partly as a bridge to risk management policy which should aim to minimize exposures to the greatest extent possible.



1.0 INTRODUCTION

1.1 OBJECTIVES OF THE CRITERIA DOCUMENT

Multimedia assessment and regulation of lead are considered a priority because of recent scientific information on environmental exposures and the related, significant implications for public health. Integrated prevention and control strategies are indicated for lead.

Since the mid-1980's, a substantial body of new information has emerged on the health effects of lead and on the environmental levels associated with such effects. In particular, there is considerable literature on the relationship between lead exposure and early neurobehavioural and developmental outcomes in young children. The result has been an evolving decline in the level of concern for blood lead in human populations.

The criteria document assembles the salient scientific information to be used in developing revised standards for lead. The document has the following objectives:

- to describe the critical health effects of lead;
- to identify and assess the dose-response information with greatest bearing on forthcoming regulatory decisions on lead;
- to provide an integrated understanding of the assessment of the risk to the Ontario population from lead exposure;
- to assess the latest scientific information relevant to decisions on existing lead standards and objectives in air, water, soil and dust, including possible revisions;
- to provide guidance on what information should be used for risk assessment of site-specific contamination and remedial scenarios; and
- to set a comprehensive scientific foundation for a consistent and coordinated approach to lead-related issues across different MOEE program areas.

1.2 THE MULTIMEDIA APPROACH TO RISK ASSESSMENT

The multimedia approach was chosen to develop the scientific criteria for lead standards. This approach considers total exposure from all environmental media, recognizing that lead is present simultaneously in food, air, water, consumer products, soil and dust. Traditionally, assessment and control of health risks have focused on a single medium, such as lead in outdoor air, usually the medium where the initial environmental release took place. This may greatly underestimate the actual environmental exposure and the risk to health of a given population.

The multimedia approach has three major advantages over the traditional single medium approach. First, it ensures that total exposure is kept below maximum allowable intake or exposure levels. Secondly, consistent risk evaluations and decisions on negligible risk can be made for each exposure pathway. Finally, it allows for a single comprehensive scientific evaluation, thus avoiding duplication of effort.

Regulation of lead requires the development of quantitative guidelines and standards. The multimedia approach allows for the derivation, in concert, of a set of single medium standards based on consideration of all exposure pathways, both direct and indirect, and utilizing a common quantitative analysis of adverse health outcomes. The intent of the "suite" of standards thus developed will protect both the environment and the community health of Ontarians.

Since 1980, there have been dramatic shifts in the levels and patterns of lead exposure for children and adults. Current estimates of the relative contribution of different media to exposure are presented in the criteria document, together with relevant indirect exposure scenarios. Incremental exposures from specific pathways, such as drinking water, are considered, as well as total integrated estimates of intake. In this way, specific exposures may be targeted for reduction in order to provide the greatest overall benefit or protection.

The scientific foundation for the application of multimedia principles to the lead question is based upon a risk assessment framework outlined in Figure 1, where risk assessment is defined.

Risk assessment is the scientific evaluation of the probability of adverse consequences (either direct or indirect), and the accompanying uncertainties, to the environment and to human health, caused by physical, chemical or biological disturbances to the environment.

Phase 1 - Hazard Identification

Hazard identification is the preliminary identification of the potentially adverse environmental and health impacts of a physical, chemical or biological disturbance to the environment. It includes ascribing a level of potential concern to the impacts, such as identifying the most sensitive plant or animal species or the most sensitive toxic endpoint upon exposure.

Phase 2 - Dose-Response Assessment

Dose-response assessment is the determination of the relationship between the magnitude of exposure from different exposure routes and the probability of the occurrence of environmental or health effects. It encompasses an assessment of the uncertainties associated with this determination.

Phase 3 - Exposure Assessment

Exposure assessment is the qualitative and quantitative determination, or estimation, of the magnitude, frequency, duration and route of exposure of a particular physical, chemical or biological disturbance to the environment. It delineates the major pathways of exposure (e.g., air, water, food); the levels of exposure from each pathway; and the total exposure of the given population from all pathways that contribute to the health risk of concern. Data for exposure assessment may be obtained from monitoring studies of the contaminant and from dynamic modelling of its environmental fate.

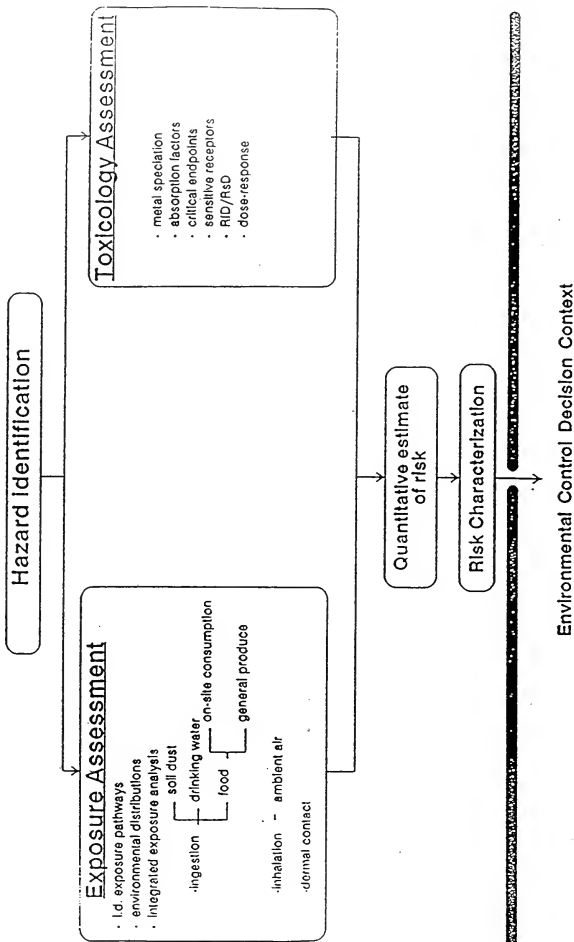
Exposure assessment extends to an evaluation of the uncertainties associated with the determination or estimation. Individual exposures to single environmental media should be evaluated in the context of an integrated multimedia assessment of total exposure.

Phase 4 - Risk Characterization

Risk characterization is the integration of the exposure and dose-response assessment to provide a description of the nature and magnitude of the risk and of the associated uncertainties. It includes the evaluation of the contribution from different exposure routes and media to the overall risk.

FIGURE 1.1

RISK ASSESSMENT FRAMEWORK



This scientific criteria document reviews and summarizes the available information under each of the four phases of risk assessment. The latter process means scientific risk assessment and does not address the social, economic, technically feasible, regulatory or legal aspects of the environmental management of lead.

The various appendices contain a compilation of Ontario information and monitoring data on the sources, emissions, levels and fate of lead in the environment. A review of the physical/chemical properties of lead is provided as well as a review of analytical methods. This information has application to the exposure assessment as well as to various aspects of the risk management phase of standards setting. This diverse information also provides a more detailed sense of the state of the environment regarding lead.

2.0 HUMAN HEALTH EFFECTS OF LEAD

Lead is a toxin with demonstrated cumulative, multi-system effects in humans. The health effects of overt lead poisoning secondary to grossly elevated blood lead levels are well known and involve several organ systems, in particular the central and peripheral nervous systems, hematopoietic system, renal, gastrointestinal system and reproductive system. More relevant to the intent of this document is a description of the health effects of lower levels of lead exposure as these are the effects of more relevance to normal environmental exposures. The fetus, infant and child under six years of age are most susceptible to the adverse effects of lead on health; pregnant women are also in a higher-risk group as a surrogate for fetal exposure. These groups, therefore, define the sensitive population segments for lead exposure.

The development of effective regulatory strategies aimed at providing an adequate level of public health protection against the toxic consequences of lead is very much related to the extent and quality of scientific information concerning low level exposure for various population subgroups. Lead is the most studied of any toxic substance. It is well recognized that the toxicological database regarding lead is massive and has been the subject of a very large number of reviews and scientific interpretations. Because of the large number of studies involved, even in the area of neurobehavioural effects in children, it is not possible nor it is the goal of this review to describe all of this information. However, it does attempt to capture the essential elements and provide a synthesis of the most current and salient findings. For more detailed discussions, the reader should refer to the critical reviews of Smith (1989), Grant and Davis (1989), ATSDR (1988, 1991), USEPA (1986a,b), USEPA (1989a,b), Needleman (1992), Harvey, (1986), Nearing (1987) and Marcus (1986) from which this review has drawn. Scientific studies concerning the effects of prenatal and early childhood exposure to lead on the neurobehavioural development of young children forms an integral component of the database. Therefore, these studies are discussed most extensively. Similarly because the questions of lead carcinogenicity, cardiovascular and reproductive effects have been other foci of research and analysis, some discussion of knowledge regarding these effects are discussed. A discussion of the quantitative aspects of dose-response information is provided in Chapter 3.

In the following sections, the use of the term "low dose lead exposure" refers to levels of lead exposure below those associated with clinical lead poisoning. Blood lead is generally expressed in $\mu\text{g}/\text{dL}$. Provided in Appendix J is a conversion chart for use when other units are specified.

2.1 METABOLIC PROPERTIES

2.1.1 Absorption

Lead may be absorbed by the body through inhalation, ingestion, dermal contact (mainly as a result of occupational exposure) or transfer to the fetus via the placenta (Goyer *et al.*, 1990). The absorption of inorganic lead through the skin (i.e. dermal route) is minimal but organolead compounds may be absorbed by this route.

In adults, under normal nutritional conditions, approximately 10% of ingested lead is absorbed into the body (USEPA, 1986a; O'Flaherty *et al.*, 1982). Young children absorb from 40% to 53% of the lead ingested through food (Alexander, 1973; Ziegler *et al.*, 1978). For lead in soil or dust, the gastrointestinal absorption rate in children has been estimated to be 30% (Drill *et al.*, 1979). Animal experiments indicate that lead of variable chemical forms in soil and dust is as available for absorption as lead in food (Dacre and Ter Haar, 1977) and *in vitro* studies have shown that conditions of acidity of the human stomach are sufficient to extensively solubilize lead assimilated from soil and dust. Bioavailability of lead from various matrices/media is therefore unlikely to vary significantly (i.e. greater than 2 fold) in children under the same gastric conditions.

Factors such as age, nutritional status and diet can influence the absorption of lead from the gastrointestinal tract. Absorption of lead is greatly increased after fasting and when the dietary intake of calcium and phosphorus is low (Blake *et al.*, 1983); lead absorption is also believed to be increased when dietary iron intake is deficient (Hryhorczuk *et al.*, 1985). The relationship between levels of lead in the blood of children and adults versus the concentration of lead in drinking water and in food appears to be basically curvilinear, with the curve almost linear at low lead levels (Moore *et al.*, 1982a; Sherlock *et al.*, 1982; Sherlock and Quinn, 1986; HMSO, 1983).

The amount of airborne lead deposited and absorbed in the lungs of adults ranges from 30% to 50% (USEPA, 1988; O'Flaherty *et al.*, 1988). No data for absorption following inhalation in children is available, however their respiratory uptake of lead is likely to be comparatively greater on a body weight basis. The rate at which lead is absorbed from the lungs is directly dependent on the rate of deposition of the lead particles in the respiratory tract. Although respiratory absorption has been thought to depend on the solubility of the inhaled lead compounds, Morrow *et al.*, (1980) have suggested that once deposited in the lungs of humans, lead particles are rapidly and completely absorbed regardless of apparent solubility, possibly due to chemical reactions *in vivo*.

Placental transfer of lead occurs in humans as early as the twelfth week of gestation, and uptake of lead by the fetus continues throughout development (Baltrop, 1969; Goyer, 1990). The concentration of lead in umbilical cord blood is correlated with maternal blood lead levels in ratios that range from 0.6 to 1.0 (Moore *et al.*, 1982; Alexander and Delves, 1981, Gershanik *et al.*, 1974; Lacey *et al.*, 1985; Koren *et al.*, 1990). In a recent study of 95 Toronto

mother-infant pairs a correlation of 0.59 ($p < 0.0001$) has been observed (Koren *et al.*, 1990). Maternal and fetal blood lead levels are almost identical and lead has been measured in the fetal brain as early as the end of the first trimester of pregnancy. The ratio of blood lead in the fetus to that of the mother is also about 0.8 to 1.0 (Gershanik *et al.*, 1974; Taylor, 1986).

2.1.2 Distribution and Retention

In the body, lead is distributed in a multi-compartmental fashion. Once absorbed, lead enters either a "rapid turnover" biological pool with distribution to the soft tissues (i.e. blood, liver, lung, spleen, kidney and bone marrow) or a "slow turnover" pool with distribution mainly to the skeleton (Ludwig, 1977; Rabinowitz, 1991).

In excess of 99% of blood lead is found within the red cells. Lead in soft tissues exchanges freely with the plasma component of lead in blood. Lead accumulation over time occurs in the body tissues, in particular the renal cortex and bone. In adults, approximately 80% to 95%, and in children about 73% of the total body lead accumulates in the skeleton (Alessio and Foa, 1983; Marcus, 1985). Bone lead is not an inert depot and it is recognized that at least one skeletal compartment of lead is readily exchangeable (Rosen, 1985). This compartment serves as a continuing internal source of lead available to other soft tissue sites, even after lead exposure is discontinued.

The biological half-life of lead in blood is approximately 16 days (Chamberlain *et al.*, 1978) and in bones about 17 years (Heard and Chamberlain, 1984). Because of the distribution kinetics of lead, its actual biological half-life is difficult to define. The biological half-life of lead in red cells has been estimated to be 25 days based on isotope studies (Rosen, 1985). As discussed by Hryhorczuk *et al.* (1985, cited by Nearing, 1987), the measured and therefore apparent half-life of lead in blood in non-isotope studies may range up to several hundreds or thousands of days, depending on age, length of exposure and period of follow-up. This reflects the multi-compartmental distribution kinetics of lead which allows constant interchange between lead in blood and lead in other body lead pools. Available evidence suggests that children retain a significantly higher relative amount of lead than adults (USEPA, 1988). O'Flaherty *et al.* (1982) demonstrated a positive correlation between length of exposure to lead and the apparent half-life of lead in blood. This also reflects the multi-compartmental distribution kinetics of lead which allows for an increased body lead burden, particularly in bone, with increased length and level of exposure.

Although blood lead concentrations reflect only recent intake (over about 40 days preceding the sampling date), under conditions of extended chronic exposure there is a steady-state distribution of lead between various organs and systems (USEPA, 1986a; O'Flaherty *et al.*, 1982). The blood lead concentration is, therefore, a reasonably good indicator of exposure from all sources and is commonly used for this purpose.

2.1.3 Excretion

The predominant mode of absorbed lead excretion is renal involving glomerular filtration and tubular excretion. Excretion through the gastrointestinal tract also occurs either directly (unabsorbed ingested lead) or via biliary clearance.

With respect to excretion of lead in breast milk, Wolff (1983) reviewed a number of studies and reported a range of mean lead concentrations in normal human breast milk from 2 to 30 µg/L. Although lead transfer from blood to breast milk occurs, it appears that the partition of lead to milk is impeded, perhaps by the affinity of lead for hemoglobin or by the lower solubility of lead in the fatty components of breast milk.

2.2 HEALTH EFFECTS OF LEAD

2.2.1 Neurological and Developmental Effects

The neurological effects of inorganic lead exposure have been widely researched. Lead can affect both the central and peripheral nervous systems. Overt neurobehavioural signs of acute lead intoxication include dullness, restlessness, irritability, short attention span, headaches, muscle tremor, hallucinations and loss of memory, with encephalopathy occurring at blood lead levels of 100 to 120 µg/dL in adults (Kehoe, 1961; Smith *et al.*, 1983) and 80 to 100 µg/dL in children (Chisolm, 1965; USEPA, 1977).

Signs of chronic lead intoxication include tiredness, sleeplessness, irritability, headaches, joint pain and gastrointestinal symptoms. These may appear in adults with blood lead levels of 50 to 80 µg/dL (Hanninen *et al.*, 1979; Lilis *et al.*, 1977). Following one or two years of exposure in an occupational setting, muscle weakness, gastrointestinal symptoms, lower scores on psychometric tests, disturbances in mood and symptoms of peripheral neuropathy were observed in adults with blood lead levels of 40 to 60 µg/dL (Baker *et al.*, 1979, 1984; Zimmerman-Tansella *et al.*, 1983; Campara *et al.*, 1984).

Frank peripheral neuropathy was observed in 14 children with blood lead levels of 60-80 µg/dL (Imbus *et al.*, 1978). Five of these children also had sickle cell disease. The authors suggested that children with sickle cell disease may have increased susceptibility to lead neuropathy.

Changes in neurophysiological function, in particular significant slowing of peripheral nerve conduction velocities, have been recorded in individual adults with blood lead levels in the range of 30 to 50 µg/dL (Seppalainen *et al.*, 1983). Araki *et al.* (1980) reported that the median motor neuron conduction velocity of adults occupationally exposed to lead (mean blood lead level 52 µg/dL) improved significantly when their blood lead was lowered following chelation of the lead by calcium disodium EDTA.

Disagreement exists as to whether these latter neurological changes represent adverse health effects. Dose-response relationships for neurological effects in otherwise asymptomatic adults have been demonstrated. From one perspective, the changes may reasonably be interpreted as adverse health effects. They represent in effect an organ system dysfunction and may be considered signs of an early subclinical stage of disease. The neurological effects have not been demonstrated to be reversible. They may also potentially enhance human susceptibility to the deleterious effects of other environmental influences.

2.2.1.1 Cross-Sectional Studies of Neurobehavioural Effects in Children

The initial research studies of the effects of low levels of lead in children were of the cross-sectional type, examining a group or groups of children at a single point in time. These studies were important in establishing an association between lead at low levels and developmental outcomes such as intelligence. These studies were undertaken in different countries, examined cohorts with different blood lead levels, used various measures of lead (blood, dentine) and investigated various health outcomes. Since the mid 1980's, there have been reports on a number of well-designed and carefully conducted cross-sectional and retrospective studies in various countries (Fulton *et al.*, 1987; Fergusson *et al.*, 1988a,b; Winneke *et al.*, 1990; Needleman *et al.*, 1990; Lynbye *et al.*, 1990; Hawk *et al.*, 1986; Schroeder *et al.*, 1985). In summarizing the general pattern of results provided, the Centers for Disease Control (CDC) has indicated that although inconsistencies can be found in the findings of individual studies, "the weight of evidence supports the hypothesis that decrements in children's cognition are evident at blood lead levels below 25 µg/dL" and that no clear threshold for a lead-IQ relationship is discernable from these data. The results of a number of these studies are briefly described below (CDC, 1991).

Fulton *et al.* (1987) investigated the influence of blood-lead on children's ability and attainment in a sample of 501 boys and girls aged 6-9 years from primary schools in Edinburgh, Scotland. Edinburgh was chosen because of its plumbosolvent water and its high proportion of houses with lead plumbing. The geometric mean blood lead level in a study subsample was 11.5 µg/dL, ranging from 3.3 to 34.0 µg/dL. Multiple regression analysis showed a small (up to 5.4 points) but significant effect on the performance in ability and attainment tests, as measured using the British Ability Scales (BAS). There was a negative relation between log blood-lead and BAS combined score, number skills, and word reading when thirty-three possible confounding variables were taken into account. The strongest relation was with reading score. The effect of lead was considered small in relation to other variables (parental socio-economic strata (SES) and intelligence, birth characteristics, home and family environment, etc.) For the BASC score, only 0.9% of a total of 45.5% variance explained by the covariates in the optimal regression model can be attributed to the effect of lead. The data demonstrated a dose-response relationship across the range of 5.6-22.1 µg/dL, with no evidence of threshold.

What is now considered a pioneering study in this area was conducted by Needleman *et al.*, (1979, 1985). 58 six- and seven-year-old children with high lead levels in their dentine (>20 ppm, associated with blood lead levels of $35.5 \pm 10.1 \mu\text{g/dL}$, the 90th percentile of a group of about 2100 children) performed significantly less well than did 100 children from the low lead level group (the 10th percentile of the same group, with <10 ppm lead in their dentine, associated with a mean blood lead level of $23.8 \pm 6.0 \mu\text{g/dL}$) on the Wechsler Intelligence Test and on other visual and auditory tests as well as on most components of behavioural assessments performed by their teachers. There was a significant decrease of 4 points ($p < 0.03$) and a uniform downward shift in IQ scores. A child in the group with high levels of lead in his or her dentine was three times more likely to have an intelligence quotient (IQ) of 80 or lower than a child in the low lead group. After further analysis using multiple regression rather than covariance analysis, and using fathers' education instead of fathers' socioeconomic status as a covariable, the test results were still determined to be significantly different (USEPA, 1983). However, in a USEPA review, these adverse effects on the nervous system were claimed to be statistically significant only for children with the highest lead levels in their dentine, reflecting blood lead levels of greater than $40 \mu\text{g/dL}$ (USEPA, 1986a, 1989a,b).

Results from studies on German children (Winneke and Kramer, 1984; Winneke *et al.*, 1982, 1983) were similar to those of Smith *et al.* in that lead caused some effect on behaviour and IQ, however the difference was only of borderline significance ($p < 0.10$) and blood lead levels accounted for only 2% of the variance in results between the groups studied.

Hatzakis and co-workers (1987) assessed the association between blood lead and IQ in 509 children living in proximity to a lead smelter in Lavrion, Greece. The blood lead levels ranged from $7.4 - 63.9 \mu\text{g/dL}$ with a mean of $23.7 \mu\text{g/dL}$. After controlling for confounding variables, including parental IQ, the findings suggested that neurobehavioural effects attributable to lead are observed at blood lead levels less than $25.0 \mu\text{g/dL}$.

Smith and co-workers (1983) investigated relationships between lead levels in teeth and measures of intelligence (Wechsler Intelligence Scale) and behaviour in over 400 British children in the Southampton survey. After correcting for social class, home quality and other confounding variables, the association between tooth-lead levels and Intelligence or academic performance was not statistically significant, although a decreasing trend was apparent. In a more recent reanalysis of this data (Pocock *et al.*, 1987), the findings are that maternal IQ had the greatest influence on child IQ, and that there was no overall evidence that tooth lead concentrations were related to child IQ once other factors were taken into account. Several other factors reflecting family circumstance, i.e. quality of marital relationship, family size and social class, were also shown to be important influences on child IQ. There was, however, a significant interaction between tooth lead concentrations and the sex of the child indicates that the lead-IQ association appears much more pronounced in boys.

In contrast to the above studies, a series of carefully designed and well-conducted studies on a total of 866 British children with blood lead levels between 4 and 32 $\mu\text{g}/\text{dL}$ showed no significant associations between lead exposure and indices of intelligence and behaviour, after socioeconomic and family characteristics were taken into account (Lansdown *et al.*, 1986; Harvey *et al.*, 1984; Harvey 1986). In accounting for the negative results compared to results from an earlier study (Yule *et al.*, 1981), Lansdown *et al.* pointed out that lead may have a noticeable effect only when other factors leading to disadvantage are present, particularly socioeconomic and home-environment factors.

The World Health Organization (WHO), Regional Office of Europe (WHO/EURO), together with the Commission of the European Communities (CEC) organized a collaborative multicenter study in which results from several independent cross-sectional studies were combined on the basis of common study protocol and quality assurance elements. The study was performed and evaluated between 1984 and 1989 and included participants from eight European countries (Winneke *et al.*, 1990). Taken together, one thousand eight hundred and seventy-nine school-aged children between age 6 and 11 were examined. Blood lead concentrations covered the range from below 5 $\mu\text{g}/\text{dL}$ to approximately 60 $\mu\text{g}/\text{dL}$. The behavioural endpoints evaluated were the WISC (4 subsets) for psychometric intelligence, the Bender Gestalt Test (GFT version) and Trail-Making test for visual-motor integration, the Vienna Reaction Device (VRD) and a delay RT task for reaction performance, and the Needleman scales for behavioural rating. According to the authors, within this blood lead range, the study confirmed that there are detectable exposure-related neurobehavioural effects in school-age children. The strongest and most consistent effects were observed in clinical tests of visual-motor integration and reaction performance. The degree of association was significant or highly significant, although the variance explained by PbB never exceeded 0.8%. Psychometric intelligence (WISC) was also affected (by association) by lead exposure, although effects were inconsistent across studies and the degree of association was borderline. The spectrum of effects seen was largely consistent with that found in previous cross-sectional research.

Winneke *et al.* (1990) provided a risk assessment of these findings based on measures of visual-motor integration and of cognitive capacity. Using averages and observed standard deviations, the percentage of children exceeding statistical criteria of deficit (i.e. 1 or 2 S.D. above or below the respective mean values) was calculated. Increasing blood lead levels from a low 5 $\mu\text{g}/\text{dL}$ to a level of 20 $\mu\text{g}/\text{dL}$, increases the proportion of children in the intermediate risk category (+ 1 S.D.) by 2.4% (WISC), 3.9 % (GFT-N) or 4.7% (GFT-I). It was noted that the full data set did not allow for the identification of an effect threshold. The relationship between blood-lead concentration and visual motor integration was considered linear.

2.2.1.2 Meta-analysis of Cross-sectional Studies

The general approach to the review of lead at low doses in relation to psychometric intelligence outcomes typically involves narrative descriptions of individual studies, which

have differed in their evaluation of essentially the same evidence. This in part stems from the inherent limitation of evaluating each study in isolation without a systematic synthesis of all this information. In order to examine this problem from a different perspective, the method of meta-analysis has been applied (Schwartz *et al.*, 1985; Needleman and Bellinger, 1989; Needleman and Gastonis, 1990). This method essentially treats individual studies as data points in a larger analysis, with summary measures from each study pooled and quantitative inferences drawn about individual research questions.

In the most recent of these assessments, 24 studies were identified which related IQ to childhood lead exposures (Needleman and Gastonis, 1990). Of these, 12 were selected for the meta-analysis on the criteria that multiple regression analysis was employed with IQ as the dependant variable and lead as the main effect, with control of non-lead covariates. The studies were grouped according to tissue type sampled. The joint P values for the blood lead studies were <0.0001 for two statistical methods, while for the tooth lead studies they were 0.0005 and 0.004 respectively. Earlier analysis of 14 studies by Needleman and Bellinger (1989) found a joint probability of the effects occurring by chance of 3×10^{-12} . The overall evidence converge strongly on the conclusion that lead at low doses is neurotoxic and that a strong link exists between low dose lead exposure and intellectual deficit in children.

2.2.1.3 Prospective Epidemiological Studies of Mental Development in Children

Earlier cross-sectional epidemiological studies of either general population or groups in the vicinity of lead smelters have provided important information on the health effects of environmental lead. These studies have produced a variety of results and each has its particular methodological limitations. Inconsistencies in results may also arise in studies because many other coexistent confounding factors also influence the development of children; the relatively small effects of lead on outcomes as compared to other possible influences; there is uncertainty regarding selection and measurement of various outcomes; and the measurement of prior exposure is tenuous.

To overcome some of these difficulties, a number of well-designed and conducted prospective studies of large groups of children have been underway. The key advantage over the cross-sectional approach is that it allows a more precise characterization of history of lead exposure during the period of development (Grant and Davis, 1989). The principal studies which have to date yielded results for analysis are those in Boston, Cincinnati and Cleveland in the United States and Port Pirie, Australia. According to Davis and Svendsgaard (1987) and reiterated in the ATSDR (1988) Report to Congress, the quality of epidemiological design is such that the evidence yielded will be superior to that in traditional single studies. These design characteristics include: 1.) use of study populations in the hundreds, thereby increasing statistical power to detect subtle effects; 2.) use of accepted, sophisticated methodologies and information exchange among principal investigators, resulting in greater uniformity in approach; 3.) appropriate statistical control of many covariates and

confounders; 4.) assessment of developmental effects in a similar fashion; and 5.) following throughout development, beginning with lead exposure measured *in utero*.

The most consistently affected endpoint has been lower scores on the Mental Development Index (MDI) of the Bayley Scales of Infant Development, which is a standardized test of infant intelligence (Mushak *et al.*, 1989). These scales are composed of three indices of mental, motor and emotional development. The Mental Development Index is designed to assess sensory-perceptual acuities and response, early acquisition of object constancy and memory, learning, and problem-solving abilities; early verbal communication; and early evidence of ability to form generalizations and classifications. The scales have a mean score of 100 and a standard deviation of 16 (Bayley, 1969).

2.2.1.3.1 Boston Study

This was the first of the longitudinal studies to be undertaken. Bellinger and colleagues (1989a, b, 1990) are following a cohort of 170 to assess the association between prenatal/early postnatal lead exposure and development. The findings thus far demonstrate that performance on the Bayley MDI at ages 6, 12, 18 and 24 months is inversely related to umbilical cord blood lead levels at birth. This inverse association appears statistically robust. MDI scores were not associated with postnatal blood lead levels (Bellinger *et al.*, 1989a). Infants with high cord blood lead levels (10-25 µg/dL) consistently scored 4 to 8 points lower than infants with cord blood lead values < 3 µg/dL. The authors indicate that the data suggest a SES effect in that the second year MDI performance of lower SES children was more greatly affected at lower blood lead levels than performance of higher SES children. It was concluded that infant vulnerability to lead's developmental toxicity appears to be greatest during the fetal period.

This same cohort has been examined for changes in performance between 24 and 57 months in relation to lead exposure and various sociodemographic factors (Bellinger *et al.*, 1991). In contrast to the infant assessments, cord blood lead level was not significantly related to children's performance on the McCarthy Scales of Children's Abilities. The Boston cohort of children did show cognitive deficits but this could not be attributed to prenatal lead. It was suggested this group appeared to have recovered from, or at least compensated for, the earlier insult. Further, the risk that a deficit would persist was increased among children with higher prenatal exposure (10-25 µg/dL), higher postnatal exposure and less favourable sociodemographic factors.

2.2.1.3.2 Cincinnati Study

Dietrich and co-workers recruited a cohort of 305 pregnant women from the inner city of Cincinnati residing in predesignated lead-hazard areas (Dietrich *et al.*, 1986, 1987a,b, 1989a,b, 1990). Blood lead concentrations of 280 newborn infants were assessed at postnatal day 10 and at quarterly periods thereafter. Blood lead concentrations for the mothers and newborns

averaged 8.0 µg/dL and 4.5 µg/dL respectively. Multiple regression analysis, which treated perinatal health factors such as birth weight and gestation as confounders, indicated independent inverse relationships between prenatal (maternal) and neonatal blood lead levels and both the Bayley MDI and PDI indices at age 3 and 6 months. Male infants from the poorest families appeared most sensitive. MDI deficits at 3 months were reported as approximately 6 points for every 10 µg/dL increment in cord blood lead and 7 MDI points per 10 µg/dl increment at 6 months. Prenatal lead levels were also related to reduced gestational age and reduced birth weight, which in turn were related to lower MDI and PDI scores. The total direct and indirect effects of prenatal lead exposure on MDI scores amounted to approximately an 8-point deficit per 10 µg/dL increase in blood lead levels (Dietrich *et al.*, 1989a).

By two years of age, no statistically significant effects of prenatal or postnatal lead exposure on neurobehavioural development could be detected. Dietrich *et al.* (1989a) have suggested that the data are consistent with a hypothesis that a postnatal neurobehavioural growth catch-up occurred in these infants fetally exposed to higher levels of lead. This neurobehavioural "catch-up response" is analogous to the phenomenon observed in infant twins compromised during prenatal development. The authors conclude that their "negative findings do not imply that lower level paediatric lead exposure is without any continuing harmful effects."

2.2.1.3.3 Cleveland Study

As in the Boston study, reports by Ernhart *et al.* (1985, 1986, 1987, 1989) and Ernhart and Morrow-Tlucate (1989) examine a cohort of children from disadvantaged urban families. For example, about half of the mothers had histories of alcohol abuse. Maternal and cord blood lead samples were obtained at time of delivery. The mean PbB for 185 mothers was 6.5 µg/dL; for 162 cord blood leads the average was 5.8 µg/dL. Of 132 mother infant pairs the correlation of blood lead levels was 0.80. A number of newborn assessments were carried out including the Brazelton Neonatal Behavioural Assessment Scale (BNAS) and portions of the Graham-Rosenblith Behavioural Examination for Newborns (G-R). Of 17 neonatal outcomes examined, only three measures were significantly related to blood lead levels. The Abnormal Reflexes and Neurological Soft Signs scales (measures of neuromuscular development for walking, standing and jitteriness/hypersensitivity respectively) showed significant increases in the amount of variance accounted for by cord blood lead; a Muscle Tonus scale showed significant effects for maternal PbB only.

With the restricted data set of 132 mother-infant pairs, only neurological soft signs were related to cord PbB. Grant and Davis (1989) and Mushak *et al.* (1989) have offered an interpretation of these results based on the report of Wolf *et al.* (1985) which indicates a statistical relationship between the Neurological Soft Signs scale and the 12-month MDI. They have suggested that one can infer that prenatal (cord) blood lead is indirectly related to the

Bayley MDI at 12 months and therefore the Cleveland finding are entirely consistent with those of the other prospective studies.

The investigators have examined the potential relationship between 6-month, 2-year and 3-year PbB to the Bayley Scales MDI and PDI at 6 months, the MDI at 1 and 2 years and the Stanford-Binet Scale of IQ (S-B IQ) at 3 years (Ernhart and Morrow-Thucate, 1989). PbB at six months was not related to any developmental outcome measure. Statistical correlation was found relating 2-year and 3-year PbB to concurrent and ensuing developmental measures (MDI and S-B IQ), however this was accounted for through the influence of the caretaking environment and was fully accounted for with statistical control of confounding variables. Therefore the results did not provide clear and consistent support for the hypothesis that low-level lead burden through age 3 is related to psychological deficit. It was concluded that the relationship of lead level and cognitive development was primarily a function of the dependence of each of these on the quality of the caretaking environment. Smaller sample size may have limited the ability to detect effects of lead, if present.

By age four years ten months, 260 children remained in the cohort. These children have now been examined to assess development just prior to school age, using the Weschler Preschool and Primary Scale of Intelligence. As with the earlier tests, effects sizes relating early lead exposures to intellectual development were small, not statistically significant and not consistent in direction.

2.2.1.3.4 Port Pirie, Australia

The Port Pirie Cohort Study is an ongoing longitudinal study of the effects of cumulative lead exposure on pregnancy outcome and childhood growth and development during the first seven years of life, among a cohort of 595 children living in the lead smelter town of Port Pirie and surrounding grain-farming districts. This group of children has been followed up from 14-20 weeks of gestation to aged 3 years (McMicheal *et al.*, 1988; Wigg *et al.*, 1988; Baghurst *et al.*, 1992a, b). The study began in 1979. Blood lead concentrations have been measured antenatally (maternal), at delivery, and postnatally at 6, 15, 24 months and annually thereafter. Systematic interview information was collected on a range of variables regarding other factors which might influence outcomes such as personal, medical and environmental factors.

The cohort of children were assessed for formal development at age 2 utilizing the Bayley Scales of Infant Development. Analysis indicated a significant negative correlation between blood lead levels at ages 6, 15, and 24 months and integrated postnatal blood lead levels, and scores on the Bayley Mental Development Scale (MDI) (Wigg *et al.*, 1988; Vimpani *et al.*, 1990). Geometric mean blood lead concentrations ($\mu\text{g/dL}$) were 14.3, 20.8 and 21.2 at the respective ages. No such association was apparent for psychomotor development. Thirteen other biological and sociodemographic variables were also found to be statistically related to Bayley scores. When multiple covariates were controlled for using multiple regression

analysis, lead levels at all postnatal ages except 24 months were found to have an independent effect on Bayley scores. This relationship was reduced but not eliminated by entering of maternal IQ or HOME (measure of home environmental quality) scores. Wigg *et al.* (1988) indicated an estimate that a child with a PbB of 30 µg/dL at age six months will have a deficit of 3.3 points (approximately 3%) on the Bayley MDI relative to a child with a PbB of 10 µg/dL. Therefore even after a range of variables with independent effects on a developmental outcome measure (e.g. MDI) have been taken into account, a small residual effect on MDI can be demonstrated.

In follow-up at the age of four years, the children's ability was evaluated with the use of the McCarthy Scales of Children's Abilities (MSCA). The blood lead concentrations at each age, particularly at two and three years, were inversely related to development at the age of four. Children with an average postnatal blood lead concentration of 1.5 µmol/L (30 µg/dL) had a general cognitive score 7.5 points lower than those with an average concentration of 0.50 µmol/L. The authors indicated that the results that increased exposure to lead results in a developmental deficit, not just developmental delay. However they caution that, because of the intrinsic difficulty in defining and controlling confounding factors when studying the relationship between blood lead levels and mental development, causal inferences must be made only with circumspection (McMicheal *et al.*, 1988).

2.2.1.4 Biological Plausibility

An important criteria to apply to the observations on effects of lead on intelligence and behaviour in non-overly lead-intoxicated children is the extent of supporting evidence of their biological plausibility both in human data and animal models. It has been suggested that subencephalopathic neurological and behavioural effects in adults, and electrophysiological evidence of effects on both the central and peripheral nervous systems in children with blood lead levels well below 30 µg/dL are supportive of plausibility (HWC, 1990). Aberrant electroencephalograph readings were significantly correlated with blood levels down to 15 µg/dL ($p < 0.05$), with effects noted at (although not significantly correlated with) blood lead levels as low as 6 µg/dL (Otto *et al.*, 1981, 1982). However, such results should be viewed with caution because the overall data is fragmentary, there is not a great deal of consistency in replication of results, and the significance of the EEG findings is unknown as normative data for children is unavailable (as reviewed in Smith, 1989). While the actual medical significance of some electrophysiological perturbations remains to be clarified and the data are insufficient to draw firm conclusions, use of evoked potential measures in paediatric lead studies may help in the establishment of a neurobehavioural threshold.

Lead-induced peripheral neuropathy which is seen in adults after prolonged exposures has been rarely observed in children. From a limited number of studies, it has been hypothesized that changes in nerve conduction velocity (NCV) may exhibit at U-shaped dose-response

function with slowing of NCV at higher blood lead levels with some increase at blood lead levels between 25 and 30 µg/dL.

Of greater significance is the possible relationship between impaired heme synthesis, hemoprotein synthesis and neurotoxicity (Moore *et al.*, 1980; Mushak *et al.*, 1989). Effects of lead on blood's biochemical function (see Section 2.2.2 below) are interrelated and can have variable biological effect. Lead-induced impairment of heme synthesis and resultant accumulation of the potential neurotoxicant d-aminolevulinate (ALA), represent a possible significant indirect impact. Subcellular CNS injury associated with lead disturbance of the heme pool can potentially effect neural mechanisms through reduction of nervous system hemoproteins available for brain cell energetics and neuronal development. Various rodent studies have demonstrated effects on the CNS at the level of protein synthesis and mitochondrial function as well as delays in cortical synaptogenesis following prenatal lead exposure (Regan *et al.*, 1989). Brain microvasculature is also a primary target for lead (Winder *et al.*, 1983). It is likely that perturbations of basic cellular function are linked to delays in functional development observed in animal toxicity studies.

Rodent and primate studies provide an assortment of observations regarding neurobehavioural perturbations of lead. Research on young primates showed that significant behavioural impairment ($p < 0.05$) of the same type as that observed in children (i.e. measures of activity, attention, short-term memory, distractibility, adaptability and learning ability) occurred when lead was administered postnatally for 29 weeks resulting in blood lead concentrations from 10.9 to 33 µg/dL (Rice, 1987). These deficits are analogous to those observed in children and were observed in monkeys at blood lead concentrations once considered the norm for children in industrialized society (Rice, 1989). Another important observation from rat studies is the wide variation in response which has been observed in lead-treated animals (Cory-Slechta *et al.*, 1989).

2.2.1.5 Persistence of Lead-induced Neurobehavioural Effects

An important question which has existed in the scientific literature regarding lead and which impacts on health policy regarding prevention of lead-induced effects is that of whether or not these subclinical effects are persistent.

With respect to neurobehavioural sequelae, it can be said generally that there is only a small amount of empirical data from which to address this question and therefore firm conclusions are difficult to state. Because the current group of longitudinal studies of neurobehavioural deficits span a limited number of years, ongoing evaluation of children from these studies would be required.

It is possible that children could compensate for certain early developmental lags through catch-up spurts in growth. On the other hand, it is generally considered that injury to the central nervous system which is caused by lead is irreversible (American Academy of

Pediatrics, 1987). Because of the sequential dependency of proper behavioural development, perturbations during early development may have longer lasting effects even if they are transient or reversible functional deficits. Further, there may also be persistence of internal lead exposure beyond early childhood in individuals carrying bone lead burdens which are mobilized later in life into other body compartments. As discussed above, the longitudinal studies in Boston and Cincinnati have indicated a persistent effect of prenatal lead exposure up to the age of one to two years.

The degree to which a neurotoxic effect is permanent will likely also be determined to some degree by whether structural damage or neurochemical change occurs, the latter being more likely associated with some degree of reversibility or compensation. Animal studies in several mammalian species demonstrate the persistence of neurobehavioural alterations well into adulthood after termination of early lead exposure. For example, an array of effects persisted into young adulthood in treated primates, after concentrations in the blood returned to 11-13 µg/dL and were maintained over the succeeding 8 to 9 years (Gilbert and Rice, 1987). Such findings are also "structurally" consistent with the long term retention/persistence of lead in neuronal and non-neuronal components of the CNS and brain of rodents after external lead exposure has ceased. Other work by Munoz *et al.*, 1986 in experimental rats also implied that early damage from lead was irreversible. Although supportive of the permanence of subclinical effects, a principle caveat with the animal work is the inability to extrapolate from doses in rats to biological measure of human body burden.

The most current and prominent study regarding persistence of effects is a follow-up study by Needleman and co-workers (1990), which reexamined 132 of 270 young adults who had been studies as children in 1975 to 1978. Impairment in neurobehavioral function was still found to be related to deciduous tooth lead content. Young persons with higher dentin levels has a markedly higher risk of dropping out and having a reading disability than lower dentin children. Higher lead levels in childhood were also associated with lower class standing in high school, increased absenteisms, lower vocabulary and grammatical reasoning scores and other functions. The authors concluded that early disturbances are associated with functional abilities later in life. An important observation was that the group of children not retested tended to have higher lead levels, suggesting that if these subjects had been retested that lead effects associations may have been greater.

In short, the question of whether subtle effects on the central nervous system from lead are irreversible and persist into later life has received limited study. Although firm conclusions cannot be reached an emerging body of animal and human evidence, together with growing knowledge regarding behavioural development, suggest that such early neurological impairments should be avoided.

2.2.1.6 Other Developmental Effects

In a study of 774 births in Port Pirie, Australia, the relative risk of premature delivery was found to increase by at least four times when maternal blood levels were greater than 14 µg/dL, compared to the risk at levels ≤ 8 µg/dL (McMicheal *et al.*, 1986). A study in Glasgow of 236 births showed that gestational age was reduced with increased umbilical cord or maternal blood lead levels. In 11 cases of premature birth (<38 weeks gestation), the maternal blood lead levels averaged ~21 µg/dL and cord blood averaged ~17 µg/dL at delivery. This is in comparison with mean maternal and cord blood lead levels of 14 µg/dL and 12 µg/dL, respectively, for the overall study (Moore *et al.*, 1982). A study based on hospital records for 4354 Boston infants did not indicate an association between maternal blood lead level and gestational age (Needleman *et al.*, 1984). After a review of the literature, the USEPA concluded that gestational age appears to be reduced as prenatal lead exposure increases, starting at blood levels as low as 15 µg/dL (USEPA, 1986b).

With respect to birth weight, a prospective study of children in Cincinnati showed a significant association between prenatal maternal blood lead levels (mean 8 µg/dL, range 1-27 µg/dL) and reduced birth weight above an apparent threshold of 12 - 13 µg/dL. A preliminary analysis of the data indicated that the decreases in birth weights ranged from 58 - 601 g per natural logarithm (ln) unit increase in blood lead level (Dietrich *et al.*, 1986; Bornschein *et al.*, 1989). However, other studies have not shown such an association. In the Glasgow study mentioned above, no association was shown between birth weight and maternal blood lead levels (mean 14 µg/dL), nor between birth weight and umbilical cord blood lead levels (mean 12 µg/dL). A study done in Cleveland showed no correlation between birth weight and maternal blood lead levels (mean 6.5 µg/dL, range 2.7 - 11.8 µg/dL) or cord blood lead levels (mean 5.8 µg/dL, range 2.6 - 14.7 µg/dL).

The Port Pirie prospective study showed a significant direct association between reduced birth weight and maternal and cord blood lead levels. The proportion of Port Pirie pregnancies producing low birth weight infants was more than twice that for births outside the town. It was noted, however, that the maternal and cord blood lead levels were actually lower in Port Pirie low-birth-weight pregnancies than when birth weights were >2.5 kg (McMicheal *et al.*, 1986). It was suggested that the fetus or placenta may have acted as a sink for maternal blood lead in the case of low-birth-weight outcomes. The increase in children's height from the ages 3 to 15 months was found to be inversely related to maternal blood lead level, in a follow-up to the Cincinnati study cited above. This was significant only in cases where the maternal blood lead level had been ≥7.7 µg/dL (Bornschein *et al.*, 1989).

In an analysis of the U.S. National Health and Nutrition Examination Survey (NHANES II) data for children 7 years old or older, controlled for four covariables including nutrition, blood lead level (n.b. current, not prenatal) was found to be a statistically significant predictor of height, weight and chest circumference. The strongest relationship was between blood lead level and height, with no indication of a threshold down to the lowest blood lead level observed, 4 µg/dL (Schwartz *et al.*, 1986).

2.2.1.7 Hearing

Lead-related decreases in hearing acuity in children have been reported (Robinson *et al.*, 1985). The probability of elevated hearing thresholds (i.e. decreased hearing acuity) in children has been found to increase significantly with increasing blood lead levels (Otto and Schwartz, 1987). The authors analyzed the NHANES II data of the audiometric results of 4,519 U.S. children. This relationship, derived single logistic regression analysis of audiometric data for both ears using frequencies of 500, 1000, 2000 and 4000 Hz (NHANES II data), applied across the entire range of blood lead levels studied, i.e. $<4 \mu\text{g/dL}$ to $>50 \mu\text{g/dL}$, with no apparent threshold. The authors concluded that a child with a blood lead level of $20 \mu\text{g/dL}$ would be 10-20% more likely to have hearing problems than a child with a blood lead level of $4 \mu\text{g/dL}$.

2.2.2 Hematological Effects

Lead intoxication adversely affects hemoglobin synthesis and red blood cell (erythrocyte) life span. The effects on the former are mediated via effects on enzymes involved in the biosynthesis of heme and of globin. The effects of lead on heme synthesis are measurable as changes in several biochemical parameters, including increased activity of the enzyme δ -aminolevulinic acid synthetase (ALAS) and decreased activities of the enzymes δ -aminolevulinic acid dehydrase (ALAD) and ferrochelatase (USEPA, 1986a). These effects often occur at blood lead levels at or below $40 \mu\text{g/dL}$ (Nearing, 1987). In children, inhibition of the activity of ALAD has been detected at blood lead concentrations as low as $5 \mu\text{g/dL}$ (Hernberg and Nikkanen, 1970; Granick *et al.*, 1973), however the health consequences of this are uncertain. It is possible that there is no ALAD-inhibition threshold level for lead (ATSDR, 1993).

Inhibition by lead of the enzyme ferrochelatase impairs the insertion of iron (II) into protoporphyrin, one of the final steps in heme formation. This impairment results in the accumulation of erythrocyte protoporphyrin (EP), either as zinc protoporphyrin (ZPP) or as free erythrocyte protoporphyrin (FEP). The threshold blood lead level (no observed adverse effect level, NOAEL) for elevation of EP in adults ranges from $20\text{-}30 \mu\text{g/dL}$. For infants and children, the NOAEL range is approximately $15\text{-}17 \mu\text{g/dL}$ (Piomelli *et al.*, 1977; 1982; Roels *et al.*, 1976; Grandjean and Lintrup, 1978; Herber, 1980; Zielhuis, 1975; Moore *et al.*, 1980).

Through its effect on heme synthesis, lead has the potential to affect biochemical processes in many organ systems. For example, in animals the heme-containing enzymes cytochrome P-450 and cytochrome C were affected, with reduced content of the former and delayed synthesis of the latter (Goldberg *et al.*, 1978; Bull *et al.*, 1979). Exposure to lead has also been shown to inhibit the formation of cytochrome P-450 in children, as reflected by the decreased activity of hepatic mixed-function oxidases (MFO). The interrelation of these biochemical

effects to adverse health effects requires clarification. The effects of lead on heme synthesis have been postulated as a mechanism for the neurotoxic effects of lead.

The reduction in erythrocyte survival time may be partially due to the inhibition by lead of the enzyme pyrimidine-5'-nucleotidase. This causes pyrimidine nucleotides, i.e. cytidine and uridine phosphates, to accumulate in erythrocytes or reticulocytes (newly-released erythrocytes). The result is that the stability of the cell membrane is compromised and cellular energetics are affected (Angle *et al.*, 1982; USEPA, 1986a). The activity of pyrimidine-5'-nucleotidase starts to decrease at blood lead levels below 5 µg/dL, however the cell contains sufficient reserves of the enzyme such that adverse effects in adults do not appear until the blood lead level reaches or exceeds 44 µg/dL (USEPA, 1986a).

One endpoint which reflects heme-related biochemical alterations is anaemia, a reduction of the hemoglobin content of blood. Anaemia results from both inhibition of heme synthesis and shortening of erythrocyte survival time (Moore *et al.*, 1980). The literature is inconclusive as to the blood lead levels at which this occurs, but suggests that reduction of hemoglobin content may occur at blood lead levels exceeding 50 µg/dL in adults and 40 µg/dL in children (WHO, 1977; Rosen *et al.*, 1974; Betts *et al.*, 1973). This reduction is not necessarily synonymous with the occurrence of a clinically observable anaemia. The LOEL for frank anaemia due to lead has been reported to be 80 µg/dL in adults and 70 µg/dL in children as reviewed in USEPA, 1986a).

2.2.3 Genotoxic Effects and Carcinogenicity

The primary concerns regarding lead have traditionally focused on adverse health effects other than cancer, although numerous investigations have examined the potential association between cancer and exposure to lead. The types of scientific information pertinent to this question includes occupational studies, long-term animal studies, short-term assays and considerations of the biochemical action of lead. These issues have been previously reviewed by the International Agency for Research in Cancer (IARC 1980, 1982, 1987) and more recently in most detail by the Carcinogenicity Assessment Group at the USEPA (USEPA, 1989c).

Lead is considered to present "genotoxic" characteristics but lead-induced gene mutations in cultured mammalian cells have only been observed at toxic concentrations. Overall it is a weak mutagen but strong mitogen. Lead has been speculated to induce renal tumours through various possible mechanisms including tumour promotion, cellular proliferation and cystic hyperplasia (Beck, 1992). Similar morphological changes in the kidney between rodent models and humans are observed. However renal adenocarcinoma is an expected outcome in rodents but relatively uncommon in humans even in large worker populations with chronic lead nephropathy.

2.2.3.1 Genotoxicity and Mutagenicity

The evidence for an effect of lead on genetic material was reviewed by IARC (1980, 1982, 1987) and was found to be conflicting. Overall studies for point mutations in bacteria systems have been negative, although it must be considered that such assays are generally considered inappropriate for assaying metal ions (USEPA, 1989c). Lead chloride, lead acetate, lead oxide and lead tetroxide were inactive in mutation tests on a number of prokaryotes and fungi including *Salmonella typhimurium* and *Saccharomyces cerevisiae*.

Lead-induced gene mutations have been observed in cultured mammalian cells but only at toxic concentrations (Hsie *et al.*, 1980; Oberley *et al.*, 1982). An exception to this is a study of V79 cell reported by Zelikoff and co-workers (1988).

Short term *in vivo* tests (dominant lethal test and chromosome aberrations in bone marrow cells) performed on mice, rats, cattle and monkeys were positive in three cases but negative in five others. The *in vivo* chromosome breaking activity of lead is affected by diet. Animals on calcium-deficient diets exhibit a higher incidence of chromosomal aberration than animals on standard diets (O'Riordan and Evans, 1974; Deknudd *et al.*, 1977).

Studies of chromosomal aberrations in workers exposed in lead-battery and lead smelter occupations performed on human cells have been carried out. The literature is inconclusive regarding the occurrence of mutagenic effects of lead exposure in humans. Cytogenic studies on humans with blood lead levels above 40 µg/dL are conflicting, with several negative reports and several positive reports describing chromatid and chromosome aberrations, breaks and gaps (IARC, 1980, 1982). For example, increased incidence of sister chromatid exchange has been observed in peripheral blood lymphocytes of workers in lead industries but not in children exposed to high levels of lead in the environment (IARC, 1987).

As with other heavy metals, the molecular mechanisms by which aberrations and mutation of genetic material occurs has not been determined. It has been suggested that lead compounds may not act directly to damage the genome, but may act instead through indirect processes, such as altering DNA replication enzymes. It has also been hypothesized in the literature that lead may be involved in the activation of the enzyme protein kinase C (PKC) (Markovac and Goldstein, 1988), a potential modulator in the transduction of external signals to cellular activations, such as cell proliferation and tumour promotion (Kikkawa and Niskizuka, 1986).

2.2.3.2 Animal Carcinogenicity

USEPA (1989c) reviewed a total of 35 studies in laboratory animals relevant to the carcinogenicity of lead. The substances tested are almost all soluble salts of lead, presumably for ease of administration. Lead acetate and subacetate have been most thoroughly investigated, with some study of lead oxide, lead phosphate, lead naphthenate, lead dimethyldithiocarbamate, and tetraethyl lead has been carried out. The potential for

carcinogenicity of lead salts has been demonstrated in rats and mice administered by the oral route, in diet, in drinking water, and via intraperitoneal and subcutaneous injection. For example, lead acetate has been shown to be carcinogenic following oral dosing in seven of nine studies with rats and one of two studies in dogs (USEPA, 1989c, p.76). In total, there is at least some evidence of the carcinogenicity of lead or lead compounds detected in 24 experiments. Although none of the available cancer bioassays were conducted utilizing inhalation exposure, the equivalent (or likely greater) bioavailability of lead through inhalation, would suggest the potential for carcinogenicity by any route.

The critical organ for oncogenic effects is the kidney, with the most characteristic response being bilateral renal carcinoma (Boyland *et al.*, 1962; Zawirska *et al.*, 1968, 1975; Azar *et al.*, 1973; Tanner and Lipsky, 1984; Nogueira, 1987; Koller *et al.*, 1985; Van esch *et al.*, 1962; Mao and Molnar, 1967; Hass *et al.*, 1965; Kasprzak *et al.*, 1985; Hiasi *et al.*, 1983; Shirai *et al.*, 1984; Van Esch and Kroes, 1969; Roe *et al.*, 1965; Zollinger, 1953; Baldwin *et al.*, 1964). Tumour induction has also been observed at other sites. Rats administered lead acetate or subacetate orally have developed cerebral gliomas (Hass *et al.*, 1965; Oyasu *et al.*, 1970). In the study by Zawirska and Medras (1968, 1975) and Zawirska (1981), increases in adrenal, testicular and prostate tumours were noted. Lead subacetate has also produced lung adenomas in strain A mice following interperitoneal injection (Stoner *et al.*, 1976; IARC, 1980).

It is important to note that in experiments on animals, tumour induction occurs only at relatively high doses of lead salts. In many cases, these doses resulted in high blood lead levels in animals and moderate to severe toxicological effects, including significant increases in mortality in all experiments (IARC, 1980). For example, renal tumours have been induced in rats exposed orally to high levels of lead acetate, lead subacetate and lead phosphate in their diet (i.e. levels of 1000 ppm or more; approximately 50 mg lead/kg body weight/day). Lead acetate also caused renal tumours in mice when administered in food at 1000 ppm (IARC, 1980, 1982).

2.2.3.3 Human Carcinogenicity

A small number of epidemiologic studies of occupationally-exposed workers are available to evaluate whether lead is causally associated with an increased occurrence of cancer (see USEPA, 1989c for detailed review). All these studies have one or more methodological limitations which preclude the definitive inference of such a causal association. Such limitations would include the lack of information on cumulative lead exposure, the known presence of other carcinogens in these workplaces (e.g. smoking, arsenic, chromium), and the general interpretive limitations of proportional mortality studies.

Malcolm and Barnett (1982) examined the mortality experience of 1898 pensioners from four lead and battery companies in England in a retrospective study spanning 1925-1976. The analysis showed no significant excess mortality from all cancers combined in any of the exposure groups. Among men dying in service, of the high exposure groups, an excess

mortality from digestive tract cancer was observed (obs. =12, PMR =167). The excesses were confined to the period 1963-1966. In a study of approximately 7000 U.S. battery plant and smelter workers, small but not statistically significant, excesses of cancers of the digestive and respiratory tract were reported (Cooper, 1976). The mean blood lead levels was 62.7 µg/dL in the battery plant workers and 79.7 µg/dL in the smelter workers. In a second follow-up study of these two cohorts statistically significant excesses of all malignant neoplasms; stomach cancer (34 observed, 20.2 expected) and respiratory cancer (116 observed, 93.5 expected) were observed in the battery plant cohort (Cooper *et al.*, 1985; Kang *et al.*, 1980). Excesses of stomach and respiratory cancer were not significant for the smelter groups. Mortality could not be related to duration of employment, as the standardized mortality ratio (SMR) decreased with years of employment. The studies lacked information on confounding variables such as smoking. A noteworthy finding was that deaths due to chronic kidney diseases were significantly elevated ($p < 0.01$) in both cohorts, although no excess of kidney cancers was observed.

Nonsignificant excesses in respiratory cancer (41 observed, 36.9 expected), bladder cancers (6 observed, 4.2 expected) and particularly kidney cancers (6 observed, 2.9 expected) were observed in a cohort of 1987 white male lead smelter workers, with at least 1 year of work at a Idaho primary lead smelter (Selevan *et al.*, 1985). All but one of the kidney cancer cases was found in the "high lead" exposure group. The SMR for chronic renal disease was also highest in this group and correlated with duration of exposure.

A non-significant increased mortality from lung cancer was observed in a small study (288 workers) of Swedish smelter workers with long-term exposure to lead (Gerhardson *et al.*, 1986). Lead poisoning did not increase the overall risk of cancer in a study of 140 deaths of male smelter workers in Australia (McMichael and Johnson, 1982). A retrospective cohort study of 4293 men employed at a British zinc-lead-cadmium smelter between 1943 and 1970 reported a statistically significant excess of lung cancer, with SMR increasing with length of employment. No smoking data were available nor was it possible to determine the contribution of simultaneous exposure to arsenic which was correlated with lead exposure and lung cancer risk (Ades and Kazantzis, 1988).

Two retrospective studies of cancer in tetraethyl lead production workers are available. In one study, comparison of mortality data of 592 tetraethyl lead workers against a non-exposed group showed that neither group exhibited excess cancer deaths (Robinson, 1974). In another study of 2510 males employed for at least 1 day at a TEL plant, the SMR for all malignant neoplasms was very slightly elevated with nonsignificant excesses of respiratory and brain cancers (Sweeney *et al.*, 1986).

Particular studies have also examined the hypothesis that paternal occupational lead exposure is a risk factor for the subsequent occurrence of Wilm's tumour in children. A case-control study of 149 Connecticut children with Wilm's tumour reported from 1935-1973 indicated that occupations of fathers of 22 tumour cases were lead related, with a resulting odds ratio of 3.7 ($p=0.0005$). This study was identified as having numerous methodological

limitations. Wilkins and Sinks (1984) studied Wilm's tumour in Ohio children, and derived an odds ratio of 1.0 for parental lead occupation versus controls, suggesting that paternal lead exposure is not a risk factor for Wilm's tumour (USEPA, 1989).

Two case reports of renal cancer in individual lead smelter workers have been reported in the medical literature (Baker *et al.*, 1980; Lilis, 1981).

In summation, all of the available studies lack quantitative exposure information, as well as information on confounding exposures associated with cancer. Cancer excesses in lung and stomach were relatively small, there is not site consistency among studies and a dose-response relationship was not demonstrated. USEPA (1989c) indicate that the epidemiological evidence for lead (and lead compounds) being a human carcinogen is considered inadequate, according to the terminology of the USEPA Guidelines for Carcinogen Risk Assessment. It was pointed out that the evidence is nevertheless thought to be "suggestive". The authors caution that although the data is not strong enough to be considered sufficient or limited evidence, it should not be interpreted as negative.

2.2.3.4 Weight-of Evidence Evaluations

Both the USEPA and IARC provide evaluations of the strength of evidence for carcinogenicity of a substance, arising from human and experimental animal data. This evidence is utilized to assign the substance to a qualitative category of hazard. A summary of these overall classifications is summarized as follows:

- USEPA (1989) Lead and Inorganic Lead Compounds
Probable Human Carcinogen (Group B2)
- IARC (1987) - Inorganic Lead - The agent is possibly carcinogenic to humans (Group 2B)

Organolead Compounds - The agent is not classifiable as to its carcinogenicity to humans (Group 3)

The basis of the USEPA classification is a combination of "sufficient" evidence from animal studies and "inadequate" evidence from human studies, together with relevant information from short-term tests and other toxic effects like nephrotoxicity.

IARC's Group 2B is similar to USEPA's Group B2 in that both classifications require "sufficient evidence in animals and "inadequate" evidence from human studies". In the 1987 supplement, IARC extended this classification to include lead and all inorganic lead compounds from the previous limit to lead subacetate, lead acetate and lead phosphate in

1980. IARC have determined organolead compounds as possessing an inadequate data base for classification.

IARC's concluded that although certain lead salts are carcinogenic in some species of animals, there is inadequate evidence that lead is carcinogenic in man (IARC, 1982). Although some mechanistic possibilities are suggested by the available animal evidence, the human epidemiologic reports suggest that lead presents a low risk of carcinogenesis in humans.

2.2.3.5 Cancer Dose-Response or Potency

The most consistent and sensitive carcinogenic endpoint in animal studies are renal tumours. In characterizing the potency of lead compounds, it would be reasonable to generally observe that the majority of the cancer bioassays were carried out with large doses. Feeding studies generally employed doses of 1000 ppm or greater in feed or drinking water to realize increased tumour incidence. At very high levels incidence was correspondingly very high. The doses of lead associated with an increased cancer incidence of 10 % in rats range approximately between 3000-30,000 mg/kg total lead over the study durations or roughly 4-40 mg/kg/day. In mice these doses were approximately equal to 7000-70,000 mg/kg over the duration of the studies or 9-90 mg/kg/day (USEPA, 1989c). Because these are relatively high doses, lead would not appear to be a potent carcinogen, relative to other carcinogens.

A key question is whether this information is sufficient to provide for an extrapolation from animal studies to a quantitative estimate of cancer potency for humans. For example, the dose-response information from the Azar *et al.* (1973) study could be subjected to low-dose extrapolative modelling, such as the linearized multistage model. However USEPA has declined to make such a cross-species extrapolation or cancer potency estimate for lead primarily because cross-species extrapolation from animal studies cannot be credibly based on animal data alone, but should include cross-species pharmacokinetic modelling to account for the differences in pharmacokinetics between humans and rodents. That there is not sufficient basis for quantitative risk assessment is a conclusion also reached by the Science Advisory Board (1989) in their review of the USEPA lead carcinogenicity assessment.

2.2.4 Blood Pressure

While for many years it has been suggested that there may be a positive correlation between blood lead concentration and hypertension, the literature was confusing on this issue as evidence was available equally to support or refute the hypothesis (see reviews by Nearing, 1987 and Sharp *et al.*, 1987). More recently, reports of well controlled studies employing relatively large population sizes, not only support this hypothesis, but also provide convincing evidence that a causal effect occurs at much lower doses than previously predicted. In the United States, analyses of the second U.S. National Health and Nutrition

Examination Survey (NHANES II, 1976-80), indicated a positive correlation between blood lead concentration and systolic and diastolic blood pressure in both men and women (Pirkle *et al.*, 1985; Harlan *et al.*, 1985). A similar relationship between blood lead concentration and systolic pressure was reported by Pocock *et al.* (1984) in the British regional heart study which examined 7735 men, age 40 to 49 from 24 British towns. A 1986 study of San Francisco bus drivers (Sharp, 1987; Sharp *et al.*, 1988) suggested a dose related correlation between both systolic and diastolic blood pressure with blood lead concentrations between 2-21 µg/dL. These authors were also the first to report smoking, caffeine intake and skin pigmentation to be confounding factors. Most recently, Sharp *et al.*, (1990), employing a cohort of 456 San Francisco bus drivers in a stringently controlled study have also reported similar results.

Analysis of data from the British Regional Heart Study showed that there was no evidence of an association between hypertension and lead-induced renal effects in men with blood lead levels below 35 µg/dL. However, the study did reveal that of 74 men 40-49 years of age with blood lead levels of >37 µg/dL, a higher proportion had systolic or diastolic hypertension than in the remaining 7661 men. These blood pressure increases were not statistically significant; however, when the results were re-analyzed using multiple regression and correcting for variation due to townsite, the association between systolic and diastolic blood pressure and blood lead level was found to be highly significant (Pocock *et al.*, 1985).

Multiple regression analysis of data derived from the NHANES survey showed a significant association between blood lead levels in the range of 7-34 µg/dL and high diastolic blood pressure in people aged 21-55 and particularly for white males aged 40-49 years ($p < 0.01$). There was no threshold apparent below which blood lead level was not significantly related to either systolic or diastolic blood pressure. Large initial incremental increases in blood pressure were found to occur at the lower blood lead levels (Pirkle *et al.*, 1985). However, the significance of these results was brought into question following another analysis of the same data using a different statistical method.

A number of smaller occupationally studies of this effect are also illustrative. In a longitudinal study of 89 Boston policemen Weiss *et al.* (1986) reported that after correcting for previous systolic blood pressure, body mass index, age, and smoking, a high concentration of blood lead was a significant predictor of increased systolic pressure. In another occupational study of 95 lead smelter workers with a mean blood lead level of 51 µg/dL, 20% had ECG abnormalities indicating cardiac ischemia. This was significant compared to the 6% incidence in a matched unexposed control population (mean blood lead level of 11 µg/dL). The lead smelter workers also displayed a slight but statistically significant increase in diastolic blood pressure, 4-5 mm Hg relative to controls. Systolic blood pressure was unaffected (Kirkby and Gynkelberg, 1985).

The mechanism of lead related hypertension remains unclear. As with many other lead toxicities, hypertension appears to be a result of multiple etiologic factors. For instance it has been suggested that lead may be competing with calcium to alter critical cell functions such as ion transport, energy production, and the function of heme-containing enzymes.

While a mounting body of evidence strongly supports a correlation between blood lead concentration and hypertension, recent studies with negative findings (Elwood *et al.*, 1988; Staessen *et al.*, 1990) cannot be ignored. Although concern exists regarding the association of blood lead level and blood pressure elevation, a dose-response relationship has not yet been clearly outlined in either animals or in humans. No threshold for this effect has been identified and the current knowledge regarding the relationship between hypertension and lead is inadequate to directly affect the setting of an upper tolerable limit of lead exposure.

2.3 OTHER EFFECTS

2.3.1 Subcellular Level Effects

No single unifying mechanism of lead toxicity across all tissues in humans and experimental animals has been demonstrated. Critical cell targets for lead toxicity are the mitochondria, and cellular/intracellular membranes. Structural mitochondrial changes occur as well as alterations in energy metabolism and ion transport. Uncoupled energy metabolism, inhibited cellular respiration and altered kinetics of intracellular calcium have been demonstrated. Several mitochondrial metabolic enzymes are inhibited by lead (e.g. ferrochelatase and mixed-function oxidases) (USEPA, 1986a; Alvares *et al.*, 1975; WHO, 1977). Additional non-mitochondrial changes include formation of lead-containing nuclear inclusion bodies and lysosomal accumulation to facilitate protein degradation in injured cells.

2.3.2 Vitamin D Metabolism Effects

Effects of lead on vitamin D metabolism have been demonstrated. The vitamin D - renal endocrine system plays a major role in the maintenance of extracellular and intracellular calcium homeostasis (Rasmussen and Waisman, 1983; Rosen *et al.*, 1980; Mahaffey *et al.*, 1982) bone remodelling, intestinal absorption of minerals, cell differentiation and immunoregulatory capacity (USEPA, 1986a). Since the metabolism of vitamin D to an active metabolite is dependent on its hydroxylation in the liver and then in the renal tubules by a heme-containing cytochrome P-450 enzyme system, adverse effects of lead on either kidney function or on the synthesis of heme may affect the circulating level of the active metabolite, 1,25-dihydroxy-vitamin D.

Dose-related decreases in circulating 1,25-dihydroxy-vitamin D levels were observed in children with blood lead concentrations ranging from 33-55 µg/dL. These decreases were statistically significant ($p < 0.001$) compared to the levels of 1,25-dihydroxy-vitamin D in children with blood lead concentrations of 10-26 µg/dL (Rosen *et al.*, 1980), and the levels were comparable to those observed in children with severe renal insufficiency (ATSDR, 1993).

Regression analysis indicated that significant decreases were associated over the range of blood lead concentrations from 12-120 µg/dL ($r = -0.88$), with no evidence that a threshold exists (Mahaffey *et al.*, 1982).

2.3.3 Reproductive Effects

Lead is a reproductive toxin in both sexes in sufficient doses. Gonadal dysfunction in males has been associated with blood lead levels of 40 to 50 µg/dL (Landrigan *et al.*, 1975; Wildt *et al.*, 1983); observed effects include altered spermatogenesis, decreases in fertility low semen volume, etc.

There may also be reproductive dysfunction in females exposed to lead in an occupational setting (Panova, 1972; Nordstrom *et al.*, 1979). Increased rates of spontaneous abortion and stillbirth have been associated with lead intoxication of female workers in the lead industry.

Animal studies have indicated adverse effects of lead on adult reproductive function and on offspring including effects on reproductive hormones, toxic effects on gonads and toxic or teratogenic effects on the conceptus (USEPA, 1986a, ATSDR, 1993). The majority of the evidence has been obtained from relatively high lead exposure situations. Low dose lead exposure has not been clearly associated with direct effects on the function of human reproductive organs in either sex.

2.3.4 Renal Effects

Historically, lead exposure has been associated with the development of renal disease involving both impairment of glomerular and renal tubular function. An adequate dose-response function between blood lead level and renal function has not been defined for chronic low dose lead exposure. The literature suggests that a blood lead level in the range 50-60 µg/dL maintained over time may be too high to protect adults from eventually developing clinically apparent kidney dysfunction. Blood lead levels below this range may be related to subclinical nephrotoxicity, the significance of which remains unclear (Nearing, 1987). Chronic nephropathy in adults and in children has not been detected with blood lead levels below 40 µg/dL (Campbell *et al.* 1977). The USEPA (1986a) concluded that nephropathy occurs in children only at blood lead levels >80 µg/dL, and usually exceeding 120 µg/dL based on the findings of Chisholm (1962), Chisholm *et al.* (1976) and Goyer (1978).

2.3.5 Immune and Endocrine System Effects

In animal studies, low dose lead exposure is associated with immunosuppression. In studies on humans, the data on immunological effects are inconsistent but indicate that lead may have an effect on the cellular component of the immune system. Immune system effects were

not observed in children with blood lead levels ≥ 40 $\mu\text{g}/\text{dL}$ (Riegart and Graber, 1976) or in adults working in the lead industry with blood lead levels of 25-53 $\mu\text{g}/\text{dL}$ (mean 38.4 $\mu\text{g}/\text{dL}$) (Kimber *et al.*, 1986). Lymphocyte transformation was significantly depressed in highly exposed occupational workers (Alomran and Shleamoon, 1988), but there was no effect on IgG and IgA levels. On the other hand, lead workers with blood lead levels of 21-85 $\mu\text{g}/\text{dL}$ have a significant suppression of secretory IgA levels which is a major factor in defense against gastrointestinal tract and respiratory infections (Ewers *et al.*, 1982). Effects of moderate lead exposure on thyroid function have been postulated but again the literature is inconsistent (Tuppurainen *et al.*, 1988). No effect of lead exposure on thyroid function has been found in children with blood leads of 2-77 $\mu\text{g}/\text{dL}$ (Siegal *et al.*, 1989). In general, the effects of lead on the endocrine and immune systems have been inadequately researched and any definitive conclusions would be premature (Nearing, 1987).

SUMMARY

- Lead accumulates in human tissues and affects multiple organ systems. The biological half-life of lead in blood is approximately 16 days and in bones about 17 years.
- The absorption of lead is influenced by such factors as nutritional status, age and diet content. There would appear to be little justification to make significant differentiation between gastrointestinal or inhalation absorption rates for dietary, soil or water lead. Estimates of these factors vary from 30 to 50% in soil and diet with possibly higher rates in drinking water or under conditions of fasting. The overall difference between would appear to be less than a factor of 2.
- Lead accumulation over time occurs in the body tissues, in particular the renal cortex and bone. In adults, approximately 80% to 95%, and in children about 73% of the total body lead accumulates in the skeleton. This compartment serves as a continuing internal source of lead available to other soft tissue sites throughout life, even after lead exposure is discontinued.
- The most significant targets include the central nervous, hematopoietic, cardiovascular and renal systems.
- Subclinical neurobehavioural and developmental effects are the critical effects appearing at the lowest levels of exposure. Because the central nervous system is especially vulnerable during early development, the fetus and children under the age of six years are the most sensitive populations. Young children are at greater risk of lead exposure for several reasons: they absorb lead more readily than adults; they have more hand to mouth activity; and their intake of lead on a body weight basis is greater. Placental transfer of lead occurs in humans as early as the twelfth week of gestation, and uptake of lead by the fetus continues throughout development.

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- Several recent, well-designed longitudinal epidemiological studies provide evidence that low level lead exposure during the prenatal and postnatal period is associated with neurobehavioural changes, decreased cognitive abilities, IQ (Intelligence Quotient) deficits and other developmental effects. The causal relationship has been the subject of considerable scientific debate. However, the evolving pattern and consistent findings of independent positive studies, together with biological plausibility considerations, give strong support for this assertion.
 - In the majority of cross-sectional studies of the effects of low levels of lead, there is an association between IQ performance on various test instruments and various lead measures (hair, teeth and blood). In some instances the magnitude of association is quite small. Although children generally receive a broad battery of developmental tests, the associations are most often seen with IQ scores. With some exceptions, the differences between low and high lead groups of children are seen on verbal performance measures. The association of lead with other sociodemographic factors, such as maternal IQ is consistently stronger than the association of lead with IQ deficit.
 - Electrophysiological evidence in children is fragmentary and cannot be utilized to draw firm conclusions regarding the underlying potential for subclinical effects. Further study of evoked potentials in lead exposed children may provide insight into the threshold for neurobehavioural effect. Evidence from animal studies on neurochemical and structural perturbations support biological plausibility of these effects. Studies in primates and rodents has yielded lead-induced effects on analogous neurobehavioral functions although extrapolation of these effects to humans is problematic for the reasons discussed.
 - It is not yet known whether these lead-associated effects are irreversible, but a recent study suggests the effects may persist into young adulthood. The question of whether subtle effects on the central nervous system from lead are permanent and persist into later life has received limited study. Although firm conclusions cannot be reached an emerging body of animal and human evidence, together with growing knowledge regarding behavioural development, suggest that such early neurological impairments should be avoided.
 - Currently no threshold has been defined for the neurobehavioural and developmental effects of lead. Considerable scientific evidence indicates that adverse effects may occur at blood lead levels as low as 10 µg/dL and perhaps lower. The significance of these impacts are best represented by a shift in the distribution of IQ or MDI (Mental Development Index) scores within the population, rather than as identifiable clinically adverse effects in individual children.
 - Neurobehavioural and developmental effects of lead in young children and infants are therefore identified as the most sensitive endpoint of lead toxicity in the most
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sensitive populations. Fetal exposure, and therefore pregnant women, are also groups at higher risk. Neurobehavioural and developmental effects should form the basis for derivation of exposure limits and other regulatory actions regarding lead.

- Short-term exposures may result in deposition of lead in brain and bone and subsequent exposure to lead released from these tissues may occur in the absence of environmental exposure. Also the neurobehavioural consequences of lead exposure may be linked to sensitive sequential developmental periods. There is no duration of exposure that has been identified as without effect.

- These potential risks are based on studies of children environmentally exposed to lead, not on theoretical extrapolations from laboratory animal studies or high-dose occupational exposures. Quantitative risk estimates of lead's carcinogenicity could be made, based on animal data and estimates of cancer potency. However, the sizeable uncertainty associated with such extrapolations and the extensive human database available for other health effects of lead, argue against employing such extrapolations for establishing environmental regulations.

- Based on animal tumour data, lead is a probable human carcinogen. Several independent studies have reported a high incidence of kidney tumours in rodents of both sexes when the animals were exposed to elevated doses of lead. The renal carcinomas were distinct from those produced by other substances, but they were observed at doses much higher than would be normally encountered in the environment. The mechanism of kidney tumour induction is not well understood although there is some evidence that lead exhibits "genotoxic characteristics." The general absence of tumours at low levels of exposure remains to be explained.

- There is evolving evidence, both human and animal, which is consistent with a small effect on blood pressure, particularly in middle-aged men. The human epidemiological observations present both positive and negative findings, and as such it is difficult to draw firm conclusions, particularly as they relate to the dose-response function at very low blood lead levels. No threshold for this effect is indicated by current studies. It is believed that characterization of risk for the purposes of standards development is most relevantly focused on developmental impacts in children and such derived guidelines would likely minimize this type of potential effect in adult groups.

3.0 DOSE-RESPONSE ASSESSMENT FOR LEAD EXPOSURE

3.1 INTRODUCTION

Dose-response assessment is the determination of the relationship between the magnitude of exposure from different lead exposure routes and the probability of the occurrence of environmental or health effects. It encompasses an assessment of the uncertainties associated with this determination.

Dose-response may refer to the magnitude and severity of a particular effect for an individual at a particular dose. A blood lead level (PbB) of 50 µg/dl in an individual with a blood erythrocyte protoporphyrin level of 1.2 µmol/L is a dose-response association. A dose-response relationship may also be defined for populations; for example, a 50% decrease in ALAD activity is expected in 60% of the population at a blood lead level of 20 µg/dL.

This chapter contains a quantitative analysis of the relationship between adverse health outcomes and blood lead in children, as well as a consideration of the possible external doses or intakes associated with these levels. The following points are included in the discussion:

- the absence of a discernible threshold for the neurobehavioural effects of lead and the significance of this absence;
- the critical endpoints, dose-response data and lowest observed adverse levels (LOAEL) in children and adults;
- approaches used by other jurisdictions in dealing with the question of tolerable intakes; and
- a derivation of an intake level of concern for child populations based upon LOAEL's expressed as PbB, quantitative oral intake/PbB relationships and uncertainty considerations.

3.2 DEFINITION OF CRITICAL HEALTH EFFECTS

The critical effects of lead must be identified in order to assess the risks of low level exposures to lead and to support the development of standards. From a scientific perspective, a critical endpoint is an effect measured at the lowest exposure; in other words, the effect for which the dose-response curve lies closest to the origin. It is important to note that a critical endpoint need not be a symptom or a medically defined illness. From the point of view of developing a regulatory strategy, the critical effect may be used to set allowable levels or to determine prevention and reduction techniques.

As discussed in the preceding chapter, lead has diverse and concurrent health effects but the earliest effects at lowest doses are hematopoietic and neurotoxic consequences in infants and young children. Past research in this area has demonstrated a progressive decline in the lowest exposure levels associated with the effects on the nervous system. Several recent epidemiological studies have provided evidence of neurobehavioural changes, decreased cognitive abilities, IQ deficits and other developmental effects, effects that are associated with elevated lead exposure in both the prenatal and postnatal periods. The causal relationship between lead exposure and these effects has been the subject of considerable scientific debate. However, the evolving pattern and consistent findings of independent positive studies are compelling support for a causal relationship.

The critical endpoint for lead exposure is therefore identified as deficits in neurobehavioural development in young children. Other undesirable effects in children and the fetus are also associated with the critical range of blood lead concentrations. These effects include: haematological perturbations; decreased birth weight; reduced gestational age; and other possible adverse effects on early growth and development.

For adult populations, the majority of reviews point to the critical health effect being elevated blood pressure. The potential for carcinogenic effects may also be an emerging issue but there is insufficient information to develop credible dose-response functions for these endpoints.

3.3 ABSENCE OF A DISCERNIBLE THRESHOLD FOR LEAD TOXICITY

Regulatory agencies generally use dose-response information to develop acceptable exposure levels for non-carcinogenic effects. The types of acceptable exposure levels include: acceptable daily intakes (ADI), tolerance limits and reference doses (RfD). They are usually defined as an estimate of the daily human exposure likely to be without appreciable risk of deleterious effects. The estimate is made with a certain degree of uncertainty and is assumed to apply to the entire population, including sensitive sub-populations, over a period of lifetime exposure. The levels are also based on the biological assumption that thresholds exist for certain types of toxic effects, such as renal cellular pathology.

The concept of a threshold, or a dose level below which adverse effects will not occur, is a critical assumption implicit to the development of a reference dose. This assumption would preclude development of acceptable exposure levels where:

- thresholds have not been discerned for the effects under consideration; or
- theoretical considerations suggest the absence of a threshold, as would be the case with certain chemicals such as carcinogens.

The existence or absence of a threshold, as well as the consideration of whether a threshold is demonstrable based on available scientific information, will shape the regulatory strategic options. A reference dose or ADI may serve as a "benchmark" for regulatory decisions based on potential impacts on human health. For example, if a specific exposure threshold is identified, a decision may be made to apply a limiting level or standard to an emission. The standard would be set so that emission sources would not cause or contribute to ambient concentrations exceeding the threshold level. If no threshold is identified, the approach may be to reduce risk to the lowest achievable levels by means of the best available control technology. Neither approach is mutually exclusive, as standards and risk reduction approaches can be applied concurrently.

At first, derivation of a threshold for lead would appear to be relatively straightforward, given the plethora of toxicological observations. As the body of knowledge about a given substance's toxicological properties increases, derivation of thresholds, or "safe" levels, can usually be made more definitively. However, because both biochemical and subclinical effects can be observed in the general population, the determination of levels at which such effects become truly adverse is difficult to ascertain. It also involves considerations of the criteria applied to definition of "adverse" and the public health significance of such subclinical impacts. As such, there remains considerable controversy whether lead has a cumulative effect which begins at very minute levels of exposure, or whether there are one or more thresholds above which impairment can be assessed.

A number of studies have found associations between children's PbB and ALAD activity, neurobehavioural development indices, and growth. These effects may persist through the lowest blood lead levels (PbB ranging from less than 10 to 15 µg/dL). A recent review concluded that there is little evidence of a threshold or no-effect level for the association between lead and IQ (Smith, 1989). The lack of a discernible threshold is supported by several recent reviews (ATSDR, 1988; USEPA, 1989; Grant and Davis, 1989; Yule and Rutter, 1986).

Animal studies may also be examined for the existence of a threshold for neurotoxicity. The primary value of animal studies, particularly *in vitro* studies, is to lend insight into the subcellular mechanism of lead neurotoxicity (Grant and Davis, 1989). Such studies have shown potentially deleterious perturbations in neurochemical function at *in situ* concentrations of 5 µM (103.60 µg/dL)¹ lead or lower (USEPA, 1986). Thus, at least at a molecular level, there may be essentially no threshold for these effects. However, a review of evidence derived from animal behaviour studies found no-observed effects levels in the region of 20 µg/dL following prenatal, postnatal and post-weaning exposure (Winneke, 1986). A mechanistic model for cognitive deficits at low level lead exposure has also suggested a threshold level (Reagan, 1989). The endpoint value is related to lead-induced dysfunctioning of the morphoregulator-neural cell adhesion molecule (NCAM). In mice, its

¹ 1 µM = 20.72 µg/dL

normal developmental sequence during final synaptic structuring is perturbed only when blood lead levels exceed 20 µg/dL.

Researchers have examined the evidence from animal studies and concluded that lead may interfere with several different biochemical and neurophysiological functions. Hence, there is no indication of any definite threshold below which lead has no effect. It is difficult to define a dose-response curve for humans, because biological data suggest that lead effects do not derive from the same mechanism at different body burdens. In other words, if lead has different effects at different levels, an entirely regular dose-response curve would not be expected. Furthermore, different thresholds might exist for different effects (Yule and Rutter, 1986).

Given the available scientific evidence, a definite threshold cannot be discerned for the subclinical effects of lead in young children; nor can a corresponding intake level be associated with no effect. However, it may be possible that a threshold does exist at some exposure below the range of 10 to 15 µg/dL. Biologists hold strongly to the threshold concept for non-carcinogenic effects. A theoretical basis for the absence of such a threshold remains to be established, although some investigators have developed "one-hit non-threshold models" to describe teratogenic dose-response relationships (Rai and Van Ryzin, 1985). In the case of lead, there is no widely accepted theoretical basis for the absence of a threshold for many of the effects associated with low level lead exposure. This is in some measure because fundamental neurochemical lesions or site of action of lead within the CNS have not yet been identified.

3.4 LOWEST OBSERVED EFFECTS LEVELS FOR LEAD

3.4.1 Lowest Observed Effects Levels for Children

The different health effects of lead and the associated lowest observed effect levels for each are summarized in Figure 3.1. Lowest observed effect levels for the critical endpoints of neurological, hematological and developmental outcomes are further detailed in Table 3.1.

The only detailed description of a dose-response curve in a pediatric population is for the effect of EP elevation (Piomelli *et al.*, 1982). The relationship was constructed using probit analysis and represents response at one and two standard deviations as the blood lead level rises. A threshold is identified at the intersection of the regression lines at 16.4 and 16.6 µg/dL, respectively.

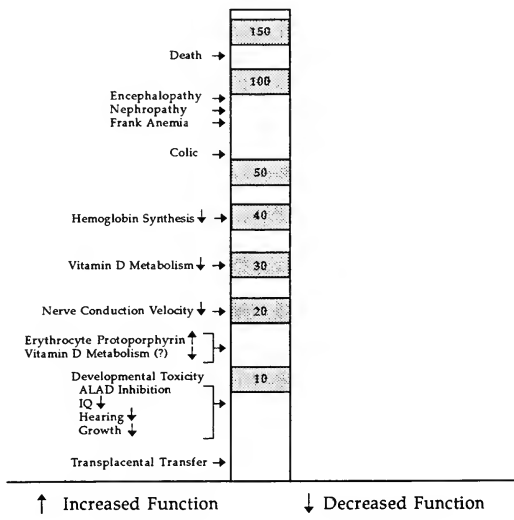


Figure 3.1 Lowest Observed Adverse Effects Level: Children
(Modified from CDC, 1991)

TABLE 3.1 LOWEST OBSERVED EFFECTS LEVEL FOR CHILDREN

LOWEST OBSERVED EFFECT LEVEL PbB (µg/dL)	NEUROLOGICAL EFFECTS	HEME SYNTHESIS AND HEMATOLOGICAL EFFECTS	OTHER EFFECTS
80-100	Encephalopathy symptoms		Acute nephropathy (aminoaciduria, etc.)
70	Peripheral neuropathies	Frank anemia	Colic, other overt gastrointestinal effects
60			Chronic nephropathy
40	CNS cognitive effects	Reduced hemoglobin; elevated CP and ALA-U	
30	Peripheral nerve dysfunction; slowed nerve conduction velocity		
25	Lower IQ, slower reaction time (cross- sectional study)		
15-20		EP elevation	Impaired Vitamin D metabolism
<10-15 (pre-natal and post-natal)	Deficits in neurobehavioural development (Bayley and McCarthy Scales); electrophysiological changes	ALAD inhibition	Reduced gestational age and birth weight; reduced size up to age 7-8 years; Py-5/-N activity inhibition

Source: USEPA, 1986; ATSDR, 1988

IQ scores are one of the more commonly employed measures of children's mental abilities. A general pattern has been suggested based on most of the available studies on the magnitude of lead effects on full-scale IQ scores (Figure 3.2) (Grant and Davis, 1989). The interpretational caveats are as follows:

- IQ is measured by different specific test instruments across the various studies; and
- the magnitude of IQ decrements reflect varying degrees of correction for confounding variables.

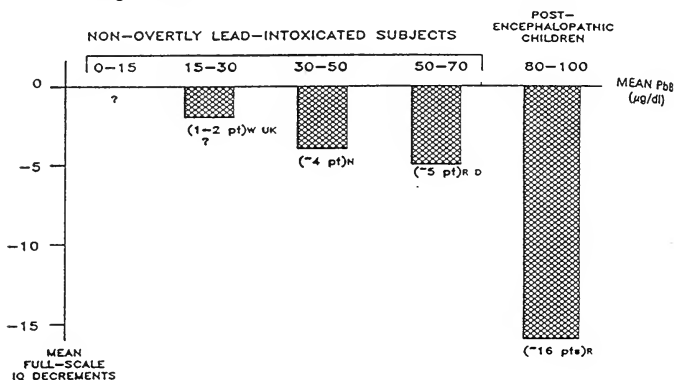


Figure 3.2 General Pattern for Effects of Lead Exposure on IQ Scores (Source: Grant and Davis, 1989)

The average decrease of approximately 5 IQ points represents a reasonable estimate of full-scale IQ decrements associated with highly elevated blood lead (mean of 50-70 µg/dL) in asymptomatic children (de la Burde and Choate, 1972; Rummo *et al.*, 1979). Average decreases of 4 IQ points were associated with tooth dentin lead levels that corresponded to blood lead values of 30 to 50 µg/dL (Needleman, 1979, 1985). The quantitative evidence for IQ deficits below 30 µg/dL PbB is inconsistent, but a difference of one to two points has been reported most often for the 15 to 30 µg/dL range. Some British studies have found no significant association at these levels after controlling for confounding variables (Harvey *et al.*, 1989; Pocock and Smith, 1989).

With respect to the critical endpoints for developmental effects, some observations can be made on the degree of deficit associated with blood lead level. The results from the prospective studies on the size of deficit in mental development scales are summarized in Table 3.2. The magnitude of the deficit is fairly consistent across all the studies: about 2 to 8 points per 10 µg/dL increment in PbB (or if assumed linear, 0.2 - 0.8 MDI points per µg/dL). The Boston study suggests that the effect occurs at PbB's as low as 10 µg/dL, with no threshold identified. The MDI difference of 4 to 8 points is the difference between high- and low-exposure groups at 6 month intervals during the first two years. Recent results from Cincinnati show an 8 point decrease per 10 µg/dL increment in blood lead. An independent analysis of the same data concluded that a threshold existed at about 12 to 13 µg/dL for reductions in birth weight related to prenatal lead exposure (Bornschein *et al.*, 1989). Other workers have made a rough estimate of a LOEL for MDI effects of 12-13 µg/dL assuming birth weight partly mediates the effect of prenatal lead on MDI (Grant, 1989).

TABLE 3.2 EFFECTS OF LEAD EXPOSURE ON MDI DEFICIT SIZE

STUDY POPULATION	AVERAGE PbB, RANGE (µg/dL)	MDI DEFICIT SIZE (POINTS)*
Boston, MA	14.6 (10-25)	4 to 8
Cincinnati, OH	~ 12.5 (7-18)	8
Cleveland, OH	? (3-15)	-
Port Pirie, Australia	~ 14 (?)	2

* Deficit per 10 µg/dL increment in PbB.

Two cross-sectional studies have investigated the possibility of a linear relationship between IQ and PbB. The first study researched a group of children of uniformly very low socio-economic status (Schroeder and Hawk, 1987). A statistically significant linear relationship was found over the blood lead range of 6 to 47 µg/dL (mean PbB of 20.8 µg/dL). The trend is shown in Figure 3.3. The second study examined the dose-response relationship of blood lead in 501 Edinburgh children against British Ability Scales (BASC) scores. The children were divided into ten groups of 50, according to increasing PbB, with group means ranging from 5.6 to 22.1 µg/dL. A difference of -5.8 points was found in the adjusted BASC scores between the first and tenth group. The data showed a dose-response relationship with no evidence of a threshold, although no quantitative relationship was cited in the study (Figure 3.4) (Fulton *et al.*, 1987).

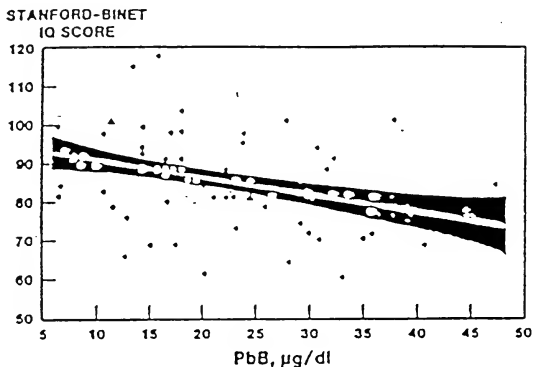


Figure 3.3 Dose-Response Relationship Between IQ and PbB in a Group of American Children (Source: Hawk *et al.*, 1986)

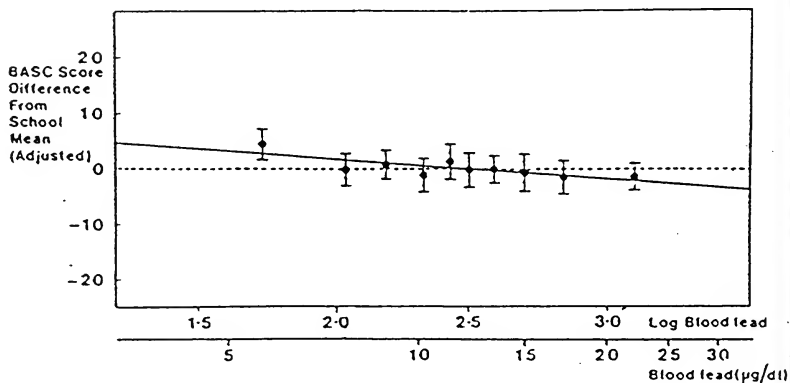


Figure 3.4 Dose-Response Relationship Between BASC Scores and PbB in Groups of Edinburgh Children (Source: Fulton *et al.*, 1987)

The findings of major prospective studies on the magnitude of the effect of PbB on other developmental measures are summarized in Table 3.3 (Grant and Davis, 1989). Analysis of the Cincinnati data indicated that gestational age was reduced by approximately 0.6 weeks for each natural log unit increase in prenatal maternal blood lead (Dietrich *et al.*, 1986). The Port Pirie study found that the relative risk of pre-term delivery (or delivery before the 37th week of pregnancy) increased 2.8 times for every 10 µg/dL increase in maternal PbB. At PbB greater than 14 µg/dL, the risk of pre-term delivery was 4.4 times that at 8 µg/dL or less (McMicheal *et al.*, 1986).

In summation, the dose-response information on either IQ measures or developmental indices appears insufficiently demarcated to develop a discrete linear dose-response relationship, although some rather crude generalizations may be possible. With respect to developmental effects, it has been suggested that prenatal exposure may be of more significance than postnatal exposure (Bellinger, 1992). In studies reporting prenatal effects the slope appears to be roughly -0.5 points per µg/dL and in those reporting postnatal effects, it appears to be -0.3 points per µg/dL. These slopes must be viewed as tentative but could perhaps be used as a crude measure of the effectiveness of various target blood lead levels in scoping the magnitude of impacts being considered.

TABLE 3.3 SUMMARY OF MAGNITUDE OF DEVELOPMENTAL EFFECTS RELATED TO BLOOD LEAD LEVEL

EPIDEMIOLOGICAL STUDY	DEVELOPMENTAL EFFECT	MAGNITUDE OF EFFECT ¹
Port Pirie	Pre-term delivery Head circumference	2.8 x Relative Risk -0.3 cm
Cincinnati	Gestational age Birth weight Birth length	-0.6 wk -225 g -2.5 cm

¹ Per µg/dL increment in PbB in Port Pirie study; per natural log unit in Cincinnati study.
Source: Grant and Davis, 1989.

3.4.2 Lowest Observed Effects Levels in Adults

The critical endpoint for adult populations is currently considered to be hypertension. Statistical analysis of the NHANES II data set demonstrated a significant linear relationship between PbB and blood pressure (Harlan *et al.*, 1985). Analysis of the survey data for 40 to 59 year old white males showed a statistically significant relationship between systolic and diastolic blood pressure and PbB's in the range of 7 to 34 µg/dL. Notably, there was no evident threshold below which the relationship was not significant. The relationship was characterized by large initial increments in blood pressure at relatively low PbB followed by

a levelling-off at high PbB. The lowest observed effect levels for health effects in adults are summarized in Table 3.4.

TABLE 3.4 SUMMARY OF LOWEST OBSERVED EFFECT LEVELS IN ADULTS

LOWEST OBSERVED EFFECT LEVEL (PbB) (µg/dL)	HEME SYNTHESIS AND HEMATOLOGIC EFFECTS	NEUROLOGICAL EFFECTS	EFFECTS ON THE KIDNEY	REPRODUCTIVE FUNCTION EFFECTS	CARDIO-VASCULAR EFFECTS
100-120		Encephalopathic signs and symptoms	Chronic nephropathy		
80	Frank anemia				
60				Reproductive effects (F)	
50	Reduced hemoglobin production	Overt subencephalopathic neurological symptoms		Altered testicular function	
40	Increased urinary ALA and elevated coproporphyrins	Peripheral nerve dysfunction (slowed nerve conduction)		Defective spermatogenesis	
30					Elevated blood pressure (M)
25-30	Erythrocyte protoporphyrin (EP) elevation (M)				
15-20	EP elevation (F)				
<10	ALAD inhibition				

Source: USEPA, 1986; ATSDR, 1988; M = male; F = female

3.5 LEVELS OF CONCERN EMPLOYED BY OTHER JURISDICTIONS

Since 1986, several governmental and international health organizations have adopted revised guidelines or standards for total lead exposure. In general, the levels have been based on health effects in the most sensitive population, young children.

In 1988, the Office of Drinking Water (USEPA) concluded that blood lead levels of 10 to 15 $\mu\text{g}/\text{dL}$ are of concern to health. A revised maximum contaminant level of 5 $\mu\text{g}/\text{L}$ was proposed for lead in drinking water, as measured in the water entering the distribution system. This translates to a lead intake of roughly 3 $\mu\text{g}/\text{day}$ (0.23 $\mu\text{g}/\text{kg}/\text{day}$) in drinking water, assuming a daily water consumption of 0.6 L/day for children (FR, 1988).

The Centers for Disease Control (CDC) position on what should be considered an elevated blood lead in children has considerable influence on the setting of levels of concern. The generally recognized level for lead toxicity has progressively declined, as more sensitive measures of toxicity and better study designs are used. Prior to 1965, a blood lead level above 60 $\mu\text{g}/\text{dL}$ was considered to be evidence of toxicity. By 1978, the defined level of toxicity had declined by 50% to 30 $\mu\text{g}/\text{dL}$. After 1985, the CDC published a revised intervention level specifying that a PbB of 25 $\mu\text{g}/\text{dL}$ or more was indicative of excessive lead absorption. In 1991, the CDC reduced its intervention level by a further 60% to 10 $\mu\text{g}/\text{dL}$ (CDC, 1991). The declining trend is largely a reflection of evolving scientific knowledge (Figure 3.5). Details of the recommended interventions at various blood lead levels are provided in Appendix H.

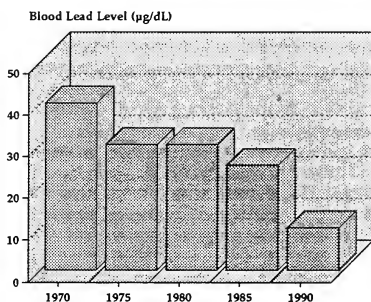


Figure 3.5 Decline in CDC Blood Lead Intervention Levels (Children)

In June 1986, the Joint Expert Committee on Food Additives of the Food and Agriculture Organization (FAO/WHO) established a provisional tolerable weekly intake (PTWI) for lead. The value was set at 25 $\mu\text{g}/\text{kg}$ body weight for infants and children (FAO/WHO 1986; WHO, 1987). The PTWI represents the permissible human weekly exposure to contaminants that are unavoidably associated with the consumption of otherwise wholesome and nutritious foods. The term "provisional" indicates the tentative nature of the guideline and

"tolerable" signifies permissible rather than acceptable. For cumulative metals like lead, the intake is on a weekly basis to allow for daily variations in intake levels, the concern being prolonged exposure. The value for lead is based on metabolic balance studies in infants. The studies indicated that blood lead was essentially maintained if daily lead intake remained below 5 µg/kg/day (Ziegler *et al.*, 1978; WHO, 1988). At lead intakes of less than 5 µg/kg/day, the mean retention was -0.48 µg/kg/day. To derive the PTWI, a small uncertainty factor of 1.4 was applied to the intake value. The smaller risk factor was justified by the high quality of the metabolic data and the fact that the most sensitive population was considered. The objective of the 25 µg/kg PTWI value was to avoid increases in blood lead concentrations.

In June 1989, the United States Food and Drug Administration (USFDA) published a proposed limit for the leaching of lead from glazes on ceramic pitchers (FR, 1989). In developing this limit, the FDA used the range of blood leads of concern in children, applying a factor of 0.16 µg/dL per microgram dietary lead per day and an uncertainty factor of 5. Fifty percent (50%) of the total lead intake in children was allocated to food. Thus, a provisional tolerable lead intake range of 6 to 18 µg/day from food was derived for a 10 kg child. By extension, this would allow for a total daily intake of 12 to 36 µg/day.

The USEPA Working Group on Reference Doses for Oral Exposures has considered it inappropriate to develop a RfD for inorganic lead. This position is based on the findings that some health effects of lead may occur at such low lead levels that they may be considered to have essentially no threshold. Such effects include changes in the levels of certain blood enzymes or deficits in children's neurobehavioural development.

In the absence of such a value, the agency has developed exposure modelling approaches which predict PbB distributions in children. Application of the models requires a number of policy decisions, including the selection of a PbB cut-off level for children. The policy decisions have not been made as yet.

3.6 DERIVATION OF LEVELS OF INTAKE OF CONCERN (IOC)

3.6.1 Calculation of Intake of Concern based on Epidemiological Data

The available epidemiological data for lead are considered a sufficient basis for the establishment of guidelines for human exposure. In deriving a reference dose or tolerable daily intake for a given chemical, the best available scientific data are reviewed to identify the highest levels of exposure that are clearly *not* associated with adverse health effects in humans. This no-observed-adverse-effect level (NOAEL) or lowest-observed-effects level (LOEL) is divided by an uncertainty factor to yield a reference dose. This factor is intended to reflect the degree of uncertainty associated with extrapolating from the NOAEL to the most sensitive sub-populations exposed as well as to provide a margin of safety.

The lack of a discernible threshold for neurobehavioural effects in children precludes the development of a traditional ADI or RfD intake. Neither intake level may be regarded as entirely safe or acceptable, because even minute exposures may be associated with a low level of risk. However, a blood lead level of concern for young children may be related to total intake in order to identify an intake of concern for individuals (IOC_{ind}). If the same principles used in the derivation of an ADI or a RfD are applied to this value, an intake of concern for populations (IOC_{pop}) may be developed to represent a maximum intake, not to be exceeded. The IOC_{pop} would represent the upper limits of intake for the average or "typical" receptor and would keep a substantially large proportion of children below the level of concern. It may be interpreted as a mean value for intake in the community against which to compare exposure estimates which provide an estimate of the central tendency or most likely exposure. Exceedance of this level may not necessarily precipitate adverse effects in an individual child. The intake level, thus derived, may then be related to lead levels in individual media for regulatory decision-making.

In most studies of lead toxicity, concentrations of lead in whole blood have been employed as the indicator of lead exposure and intake values are generally unknown. Thus, calculations are based upon blood lead as an "internal" measure of total exposure. Although blood lead measurements are not direct surrogates of dosages received at target sites, it is an index of relatively stable exposure and it can respond rapidly to variations in intake. There is an implicit assumption that blood lead reflects a steady state condition resulting from a daily intake value. The PbB values selected for derivation of the IOC_{pop} are therefore assumed to be equivalent to a lowest-observed-adverse-effect level (LOAEL).

The preceding discussion applies to LOAEL's and dose-response considerations for specific effects of lead. However, the health effects associated with various PbB's are a continuum of effects, ranging from subtle molecular perturbations, biochemical responses and cellular dysfunction to clear organ system pathologies and clinical toxicity. The overlap of PbB ranges associated with different effects, and the interrelationship of these effects, make it very difficult to determine an appropriate threshold level below which there are no significant risks of adverse effects. The severity of adverse effects increases with increasing PbB as does the involvement of a number of organ systems. However, it is uncertain at what level a single subtle effect, or aggregate of such effects, becomes sufficiently pronounced to be considered an adverse health effect. Also, biochemical changes, such as altered enzyme activities, are believed to underlie lead toxicity at the lowest measured levels and these changes show no apparent threshold on a subcellular molecular level.

In summary, there appears to be a general, quantitative relationship between PbB levels in children and various adverse outcomes, but a discrete dose-response relationship cannot be precisely established, particularly for the most subtle effects. However, there appears to be a trend of convergence of initial effects at PbB's in the range of 10 to 15 µg/dL and perhaps lower, although the available data do not indicate a clear threshold.

When deriving a PbB of concern, USEPA drew the following conclusion (USEPA, 1989):

"...a blood lead concentration of 10-15 µg/dL, and possibly lower, remains the level of concern for impaired neurobehavioural development in infants and children. Given the fact that such effects have been associated with blood lead measures in pregnant women, umbilical cords, and infants up to at least 2 years of age, there is no apparent distinction at present as to whether this level of concern applies only to fetuses or infants or preschool-age children. Thus, a PbB of 10-15 µg/dL, and possibly lower, ought to be avoided in pregnant women, fetuses, infants, and young children, although it is recognized that pregnant women *per se* are not necessarily a population at risk."

Thus, the initial observable health effects appear to occur in the PbB range of 10-15 µg/dL. On the basis of the above considerations, the appropriate LOAEL should be the lower end of the range or 10 µg/dL for infants, young children, and pregnant women as exposure surrogates for the fetus. This level allows the derivation of a corresponding intake of concern for sensitive populations. It must be stressed, however, that a level of concern is not the same as a threshold value. A level of concern represents a PbB associated with effects that may warrant a medical or regulatory intervention. It is possible that a toxicological effect or biological perturbation might occur at lower levels of exposure (Davis, 1990).

To derive a corresponding intake value, PbB must be related to total lead intake. The mathematical relationships between intake sources of lead and PbB are different for children and adults. For infants, the calculation is based upon estimates of the ratio of dietary lead intake to blood lead relationships (Ryu *et al.*, 1983, 1985). Ryu measured the approximate daily lead ingestion and the PbB of 25 formula-fed infants aged 8 to 195 days, under conditions of low prenatal exposure. During the first 112 days, 17 infants received formula from glass bottles (average daily lead intake of 17 µg). From 112 to 195 days, ten infants received homogenized milk supplied in cartons (lower lead) and 7 received milk/formula from cans (higher lead). The mean lead intake and corresponding PbB for the two exposure groups are given in Table 3.5. The trends in PbB for the groups over time are shown in Figure 3.6. USEPA used these values to derive a slope based on the differential blood lead response, as follows: (USEPA, 1986)

$$\Delta \text{PbB} = \text{PbB}_{\text{higher}} - \text{PbB}_{\text{lower}} = C(\Delta \text{intake})$$

$$7.2 \text{ µg/dL} = C (45 \text{ µg/day})$$

$$C = 0.16 \text{ µg/dL per µg/day}$$

The calculated slope should be regarded as an underestimate, because the blood leads of the infants had not reached equilibrium. It is also known that the relationship between PbB and lead uptake from any source can best be described as curvilinear: at low intake values, the

relationship is approximately linear, but the curve flattens considerably at high intake levels. It is recognized that this study may be limited by the following two factors. First, the number of children (16) studied is relatively small. Secondly, there is uncertainty whether a steady state situation existed. However, it is currently the only study to have examined the relationship between PbB and lead uptake at the lower range of blood lead concentrations and provides known intake values with respect to resultant blood lead level.

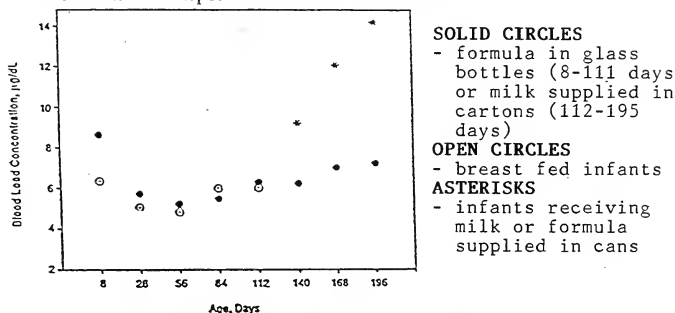


Figure 3.6 Trend in Blood Lead Levels of Infants (Source: Ryu *et al.*, 1983)

TABLE 3.5 LEAD INTAKE VALUES AND CORRESPONDING BLOOD LEAD LEVELS IN INFANTS

AGE (days)	BLOOD LEAD OF COMBINED GROUP (µg/dL)		AVERAGE LEAD INTAKE OF COMBINED GROUP (µg/day)	
8	8.9		17	
28	5.8		17	
56	5.1		17	
84	5.4		17	
112	6.1		17	
	Lower Lead Group	Higher Lead Group	Lower Lead Group	Higher Lead Group
140	6.2	9.3	16	61
160	7.0	12.1	16	61
196	7.2	14.4	16	61

Source: Ryu *et al.*, 1983; USEPA, 1986.

A more recent analysis of these data suggests a slightly steeper slope of approximately 0.24 µg/dL per µg Pb intake in milk and formula (Marcus, 1989). The analysis utilized non-linear and piecewise linear models to examine PbB-dietary slopes from the Ryu data, as well as data on Scottish infants (Lacey *et al.*, 1985) and school children exposed to lead in drinking water (Laxen *et al.*, 1987). Using the slopes and converting the water-based slopes to uptakes, three roughly consistent coefficients were derived: Ryu data, 0.24; Lacey data, 0.25; and Laxen data, 0.16 (Marcus, 1989). There were significant differences in slopes for individuals.

Two caveats of scientific evaluation of these slopes must be addressed here. First, the Ryu data are derived from infants no older than 192 days (roughly 6 months). The gastro-intestinal (GI) tract has greater absorptive capacity at very young ages and this capacity decreases as the GI tract matures. There are therefore limitations in using these slopes to derive intakes for one to four year olds, who may have lower uptake capacities. Secondly, the more recently derived slopes (USEPA, 1986; Marcus, 1989) are expressed in units of total exposure (µg/day) rather than as on a body-weight basis (µg/kg/day). The average body weight of a four to six month old infant, based on published allometric figures, is roughly seven kilograms (7 kg), whereas the average body weight of a child six months to four year old is 13 kg (HWC, 1988). However, a linear relationship between the rate of change of slope with increasing body weight cannot be assumed; in other words, the coefficient may not be 50% less at twice the body weight. In fact, even higher coefficients may be appropriate for lead uptake in young infants who ingest lead in soil or water between meals or who have nutritional deficiencies that facilitate lead absorption. The two diverging arguments would suggest as a reasonable assumption the use of a median average uptake rates of those reported for use in deriving of an intake of concern.

Therefore, for the purposes of calculation, a value of 0.21 µg/dL per µg/kg/day is selected because although the coefficient may decrease with age and reduced gut absorption, this is offset by the prudence dictated by observations on nutritional deficiency and individual variation. The total lead intake equivalent to a LOAEL of 10 µg/dL (which is also the CDC blood lead level of concern) is given by the equation:

$$\begin{aligned} \text{IOC}_{\text{ind}} &= \frac{\text{LOAEL or PbB of concern}}{\text{intake/PbB slope factor}} = \frac{10 \mu\text{g/dL PbB}}{0.21 \mu\text{g Pb per dL PbB per } \mu\text{g/kg/day}} \\ &= 47.6 \mu\text{g/day or } 3.7 \mu\text{g/kg/day for a 13 kg child (0.5-4 yrs).} \end{aligned}$$

To derive the IOC_{pop} an uncertainty factor of 2 is applied to the IOC_{ind} :

$$\begin{aligned} \text{IOC}_{\text{pop}} &= \frac{\text{IOC}_{\text{ind}}}{\text{Uncertainty Factor}} = \frac{3.7 \mu\text{g/kg/day}}{2} \\ &= 1.85 \mu\text{g/kg/day or } 24 \mu\text{g/day for an 0.5 to 4 year old child.} \end{aligned}$$

Describing the uptake of lead by the fetus relative to environmental exposures is more complex. The pregnant woman is considered to be the exposure vehicle for the fetus because lead is readily transferred across the placenta and uptake continues throughout the gestational period. *In utero* exposures may occur at highly sensitive periods in organ or organ system development. Estimating the lead intake to blood lead level relationship for pregnant women is complicated by the possible mobilization of lead from past exposures; in other words, lead which has accumulated in internal storage pools such as bone. USEPA has suggested that fetal exposures are probably dominated by maternal blood lead stores from past, higher level exposures (USEPA, 1989c). Therefore an equivalent derivation based on maternal exposures is not supportable. However, because adults generally have lower uptake coefficients for lead than children, intake at the derived IOC is not expected to contribute significantly to body burden in pregnant women.

Based on the above calculations, an average daily intake for lead of 1.85 $\mu\text{g}/\text{kg}/\text{day}$ is derived as a total intake of concern for sensitive populations within the community. This is based upon a LOAEL in infants and young children of 10 $\mu\text{g}/\text{dL}$, converted to an intake, with an applied uncertainty factor of 2.

The IOC should not be regarded as an acceptable intake or as a safe level below which no effects will occur, because a threshold for critical effects has not been scientifically established. It should be regarded as a level which, if applied to the general population, should offer some measure of protection to the majority of young children through prevention of environmental exposures which would lead to unduly elevated blood lead. It is not an intake level with no risk but rather a crude estimate of the total intake on average above which there may be some degree of health concern. Exceedance of this value may not precipitate effects in an individual child, but should be viewed as an average intake not to be exceeded in the community. This then provides the basis for health-based standards for community protection. Because the IOC is derived from oral ingestion data, this value should be applied with more caution to situations where there are very large inhalation exposures anticipated. Nevertheless, the criteria can be utilized in limiting total lead exposure (i.e. from all sources) as tracer studies (Chamberlain et al., 1978; Rabinowitz et al., 1976) show no difference in the distribution of lead to tissues whether taken up via the lung or gut.

This IOC is lower than the value of 3.5 $\mu\text{g}/\text{kg}/\text{day}$ established by the World Health Organization, the rationale for the latter being no anticipated increase in PbB. The recommended IOC is also lower than a value derived in a similar manner during the recent development of the Federal Drinking Water Objective for Lead. The differences stem from the choice of a LOAEL of 10 $\mu\text{g}/\text{dL}$ PbB rather than 15 $\mu\text{g}/\text{dL}$ and from using a revised intake conversion factor (Marcus, 1989) rather than the earlier factor derived by USEPA. However, the total intake value of 24 $\mu\text{g}/\text{day}$ does fall within the tolerable intake range of 12 to 36 $\mu\text{g}/\text{day}$ suggested by the USFDA. More importantly, it should maintain more than 99% of children at blood lead levels below 15 $\mu\text{g}/\text{dL}$, a level at which the CDC recommends individual case management.

3.6.2 Rationale for the Uncertainty Factor

Uncertainty factors have been traditionally applied in the derivation of guidelines from either animal toxicity data or epidemiological data which is limited in some manner. There is no scientific rationale which can be developed for the determination of the appropriate size of such factors. These are judgements based on the quantity and quality of data available. As well in the case of deriving an intake of concern these traditional approaches should not necessarily be applied in the same manner, as unlike an ADI or reference dose, the IOC does not define an intake below which there are no effects or zero risk.

The principle factors in selecting a value of 2 for the above derivations were:

- the LOAEL identified for low level effects of lead was based upon several studies involving several hundred young children. The data is therefore taken directly from the most sensitive receptor. A strong consensus of results and interpretation has been achieved. Several epidemiological studies have provided corroborating evidence for neurobehavioural deficits and low-level lead exposure in young children. The reasonably close agreement between these studies provides a higher degree of confidence in the LOAEL selected and decreases the uncertainty.
 - There is a relatively small uncertainty in calculating the oral intake which corresponds to the selected blood lead value. There are notable variations in the mathematical relationships derived between oral intake and PbB. The most conservative average slope value is adopted.
 - There is uncertainty in the selection of a single LOAEL value for children. The effects of lead are best described as a continuum involving multiple organ systems and having no apparent threshold for some effects. Some effects may occur at levels somewhat below the LOAEL of 10 µg/dL.
 - The toxicological endpoints associated with the LOAEL are subtle biochemical perturbations which would not be considered obvious clinical health effects and the neurobehavioural performance deficits measured at this level appear to be small on an individual basis. The significance of the neurobehavioural deficits is more appropriately measured in terms of a shift in the population average.
 - The toxicological database for lead may be the most extensive one available for any toxin, and thus, substantially less uncertainty is expected than for substances with little toxicological data.
 - The selected LOAEL of 10 µg/dL lies at the low end of the range of blood lead levels of concern for neurobehavioural deficits in individuals. The current body of information is not adequate to evaluate LOAEL's or thresholds for individual effects below 10 µg/dL.
 - Human data are used in the calculations so that an uncertainty factor for inter-species extrapolation is not required.
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SUMMARY

- The critical endpoint for lead exposure is identified as deficits in neurobehavioural development in young children. Other undesirable effects in children and the fetus are also associated with the critical range of blood lead concentrations. These effects include: haematological perturbations; decreased birth weight; reduced gestational age; and other possible adverse effects on early growth and development.
- For adult populations, the majority of reviews point to the critical health effect being elevated blood pressure. The potential for carcinogenic effects may also be an emerging issue but there is insufficient information to develop supportable dose-response functions for these endpoints.
- Taken together the body of studies indicate a general, quantitative relationship between blood lead levels in children and various adverse outcomes. A discrete dose-response relationship cannot be precisely established, particularly for the most subtle effects. There appears to be a convergence of a variety of initial effects at blood lead levels in the range of 10 to 15 µg/dL and possibly at lower levels. However, the available data do not allow identification of a clear threshold. Blood lead levels as low as 10 µg/dL in infants, children and the fetus are associated with adverse neurobehavioural and cognitive changes. This level is therefore identified as the Lowest Observed Adverse Effect Level (LOAEL) for sensitive populations.
- The health effects associated with various blood lead levels represent a continuum of effects, ranging from subtle molecular perturbations, biochemical responses and cellular dysfunction to clear organ system pathologies and clinical toxicity. The overlap of PbB ranges associated with different effects, and the interrelationship of these effects, make it very difficult to determine an appropriate threshold level below which there are no significant risks of adverse effects. The severity of adverse effects increases with increasing PbB as does the involvement of a number of organ systems. However, it is uncertain at what level a single subtle effect, or aggregate of such effects, becomes sufficiently pronounced to be considered an adverse health effect. Also, biochemical changes, such as altered enzyme activities, are believed to underlie lead toxicity at the lowest measured levels and these changes show no apparent threshold on a subcellular molecular level.
- In most studies of lead toxicity, concentrations of lead in whole blood have been employed as the indicator of lead exposure and intake values per se are generally unknown. Consideration of limiting intakes for lead are based upon blood lead as an "internal" measure of total exposure.
- In adults, effects on blood pressure have been observed across the range of blood lead levels studied, again with no evidence of a threshold.

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- Given the available scientific evidence, a definite threshold cannot be discerned for the subclinical effects of lead in young children; nor can a corresponding intake level associated with no effect. However, it may be possible that a threshold does exist at some exposure below the range of 10 to 15 $\mu\text{g}/\text{dL}$. In the case of lead, there is no widely accepted theoretical basis for the absence of a threshold for many of the effects associated with low level lead exposure.
 - In cross-sectional epidemiological studies, the evidence for IQ deficits below 30 $\mu\text{g}/\text{dL}$ PbB is inconsistent, but a difference of one to two points has been reported most often for the 15 to 30 $\mu\text{g}/\text{dL}$ range. The CDC has estimated IQ deficits of approximately 0.25 points per every microgram increment in blood lead levels.
 - The results from the prospective studies on the size of deficit in mental development suggest the magnitude of the deficit is fairly consistent across all the studies: about 2 to 8 MDI points per 10 $\mu\text{g}/\text{dL}$ increment in PbB (or if assumed linear, 0.2 - 0.8 MDI points per $\mu\text{g}/\text{dL}$).
 - Findings from the prospective studies on the magnitude of the effect of PbB on other developmental measures provide limited dose-response information. In one study, gestational age was reduced by approximately 0.6 weeks for each natural log unit increase in prenatal maternal blood lead. Another study found that the relative risk of pre-term delivery (or delivery before the 37th week of pregnancy) increased 2.8 times for every 10 $\mu\text{g}/\text{dL}$ increase in maternal PbB. At PbB greater than 14 $\mu\text{g}/\text{dL}$, the risk of pre-term delivery was 4.4 times that at 8 $\mu\text{g}/\text{dL}$ or less.
 - The dose-response information on either IQ measures or developmental indices is insufficient to develop a clear linear dose-response relationship. With respect to developmental effects, prenatal exposure may be of more significance than postnatal exposure. In studies reporting prenatal effects the slope appears to be roughly -0.5 points per $\mu\text{g}/\text{dL}$ and in those reporting postnatal effects, it appears to be -0.3 points per $\mu\text{g}/\text{dL}$. These slopes must be viewed as tentative but could perhaps be used as a crude measure of the effectiveness of various target blood lead levels in scoping the magnitude of impacts being considered.
 - The lack of a discernible threshold for neurobehavioural effects in children precludes the development of a traditional Acceptable Daily Intake (ADI) or Reference Dose (RfD) intake. Neither of these type of toxicological benchmarks may be regarded as entirely safe or acceptable in the case of lead, because even minute exposures may be associated with some degree of risk. However, the blood lead level of concern (10 $\mu\text{g}/\text{dL}$) in young children, which represents a "threshold" for U.S. and proposed Canadian intervention strategies" may be related to total intake to identify an intake of concern for individuals (IOC_{ind}). On a population, rather than an individual, basis, an intake level of concern (IOC_{pop}) is derived based upon the LOAEL of 10 $\mu\text{g}/\text{dL}$, quantitative oral intake - blood lead relationships and application of an uncertainty
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factor. The recommended intake of concern to be used in the derivation of multimedia guidelines that will be protective of sensitive populations is 1.85 $\mu\text{g}/\text{kg}/\text{day}$ or 0.0018 $\text{mg}/\text{kg}/\text{day}$. This is equivalent to a daily intake of about 24 $\mu\text{g}/\text{day}$ in a child aged 0.5 to four years. The IOC should not be regarded as an acceptable intake or as a safe level below which no effects will occur, because a threshold for critical effects has not been scientifically established. It should be regarded as a level which, if applied to the general population, should offer some measure of protection to individual children. This also provides an appropriate measure against which to compare average or typical exposure estimates, which are themselves a measure of central tendency. Exceedance of this intake may not necessarily precipitate adverse effects in an individual child, but may be viewed as a mean intake not to be exceeded in the community.

In utero exposures may occur at highly sensitive periods in organ or organ system development. Estimating the lead intake to blood lead level relationship for pregnant women is complicated by the possible mobilization of lead from past exposures; in other words, lead which has accumulated in internal storage pools such as bone. Fetal exposures are probably dominated by maternal blood lead stores from past, higher level exposures. Therefore an equivalent derivation based on maternal exposures is not supportable. However, because adults generally have lower uptake coefficients for lead than children, intake at the derived IOC_{pop} is not likely to contribute untowardly to body burden in pregnant women. The value derived here is somewhat more conservative than ADI values developed by other regulatory jurisdictions.

The principle areas of uncertainty in deriving an intake of concern are in calculating the oral intake which corresponds to the selected blood lead value. There are notable variations in the mathematical relationships derived between oral intake and PbB. The most conservative derived slope is therefore utilized in deriving the IOC. There is relatively high confidence in the overall database and choice of a LOAEL from the data in child studies.

4.0 MULTIMEDIA HUMAN EXPOSURE ASSESSMENT

4.1 INTRODUCTION

Lead is present and persistent in diverse environmental media. Adequate risk assessment therefore must consider specific lead exposures and their relative contributions to the total exposure of higher risk population groups.

A number of approaches may be used to assess the lead exposure of human populations. Broadly speaking, exposure may be assessed through:

- environmental monitoring and subsequent modelling of pathway-specific intakes, or
- population-based studies involving biological monitoring.

In the first approach, levels and trends of lead are measured or modelled in environmental media and combined with models of consumption and contact to generate age-specific estimates of intake. In the second approach, lead is measured directly in the receptor in biological media, typically blood lead (PbB) or lead in bone.

Since 1970, many epidemiological studies have been carried out in Ontario and elsewhere. Such studies explore the relationship between environmental lead levels and exposure in specific populations, the latter being represented by corresponding blood lead levels. Although most of the studies have demonstrated an association between lead levels in air, soil or house dust and blood lead, inherent limitations have made it difficult to establish quantitative relationships.

Modelling of lead exposure using environmental levels data has evolved to the extent that more than one form is available. A commonly utilized and simplistic approach involves point or deterministic estimates of intake, providing values of receptor contact in units of $\mu\text{g}/\text{day}$ or $\mu\text{g}/\text{kg}/\text{day}$. This approach is referred to as a multimedia deterministic model. Alternatively, more complex integrated pharmacokinetic-based models of exposure may be employed.

The findings of relevant epidemiological studies are described in this chapter. These provide information on the status of blood lead levels in Ontario populations and explore relationships between PbB and lead levels in specific media. A deterministic model was selected to develop media-specific estimates for lead intakes as well as integrated exposure profiles for general Ontario sub-populations. Specific exposure scenarios based on incidental exposures or high risk factors are also examined. The exposure estimates provide the basis for allocation of daily intake in the derivation of multimedia standards and regulatory strategy. Recent developments in integrated uptake biokinetic (IU/BK) modelling for lead are also discussed.

4.2 EPIDEMIOLOGICAL STUDIES OF LEAD EXPOSURE

4.2.1 Status of Blood Lead Levels in Ontario

4.2.1.1 The Ontario Blood Lead Study (1984)

In 1984, the Ontario Ministries of the Environment, Health and Labour coordinated a study to determine blood lead levels and associated risk factors in Ontario children (Duncan *et al.*, 1985). Children were tested in urban, suburban and rural areas of the province, at locations removed from known industrial point sources of lead. A total of 1269 children, aged less than six years, were tested, the majority (1155) ranging in age from four to six years.

Soil sampling and analysis were conducted by the Phytotoxicology Section of the MOEE. Soil samples (0-5 cm depth) were collected from a minimum of three locations per school yard, from unpaved play areas where children would have frequent, direct contact with soil. Soil lead levels in unseeded areas appeared to be lower than soil lead levels in adjacent grassy sites.

Urban children had the highest blood lead levels (geometric mean $12.0 \pm 4.4 \mu\text{g/Dl}$), while suburban children had higher blood lead levels ($10.0 \pm 3.5 \mu\text{g/Dl}$) than rural children ($8.9 \pm 3.9 \mu\text{g/Dl}$).

A total of 65 risk factors were assessed. Twenty per cent (20%) of the variance in the blood lead levels could be explained by the following factors: proximity to industries and gas stations; soil lead levels; traffic density; age; and socio-economic status. It is important to note that this study did not evaluate the contribution of diet, interior house dust, or lifestyle factors to elevated blood lead levels.

4.2.1.2 Blood Lead Surveys of the South Riverdale Community, Toronto (1982 - 1992)

Since 1982, the Toronto Department of Public Health has conducted annual blood lead testing of South Riverdale children under the age of six years. South Riverdale lies in the vicinity of a secondary lead smelter. The designated testing area consists of whole city blocks, or parts thereof, where soil lead levels in the past have frequently exceeded 1000 ppm. Soil lead levels were based on MOEE soil sampling surveys.

The earlier surveys had indicated that children in the designated testing area had higher blood lead levels than other urban children who did not live near a major industrial lead source. For example, in 1984, children living in South Riverdale had a mean lead level of $13.99 \pm 1.58 \mu\text{g/dL}$ (Macpherson, 1987a), as opposed to the level of $12.0 \pm 4.4 \mu\text{g/dL}$ found in urban children in the 1984 Ontario Blood Lead Study (Duncan *et al.*, 1985). In fact, more

than three times as many children tested in South Riverdale (18%) had blood lead levels exceeding the 20 µg/dL intervention level.

To determine the most significant factors associated with the observed differences in blood lead levels, a comparison was done between the 1984 South Riverdale survey and the Ontario Blood Lead Study (Macpherson, 1987a). The key findings of the comparison study are as follows:

- Children living in an area impacted by an industrial lead source had significantly higher blood lead levels than children removed from such a source.
- Children living in South Riverdale had a higher (2-3 µg/dL) mean blood lead level than did children in other urban areas of the province.
- More than 18% of the children in South Riverdale had blood lead levels exceeding the 20 µg/dL intervention level, compared to only 5% of urban children in the Ontario Blood Lead Study.
- In South Riverdale, levels of lead in residential soil (weighted geometric mean of 641 ppm) were markedly higher than the lead levels in school yard soil in other urban areas of the province (weighted geometric mean of 47 ppm).
- The air lead levels in South Riverdale exceeded the air lead level in other urban areas (0.8 µg/m³ as opposed to 0.5 µg/m³).
- Although no single environmental factor was sufficient to predict children's blood lead levels, four factors accounted for 21% of the variance:
 - immediate proximity to numerous industries;
 - neighbourhood traffic density;
 - socio-economic status of family; and
 - soil lead level in the child's immediate environment.

It is noted that the South Riverdale comparison study did not examine the influence of the following factors: diet; personal hygiene; presence of lead paint in the immediate environment; and lead levels in dust fall and house dust. Although dietary lead intake was not evaluated in either this study or the Ontario Blood Lead Study, no significant difference is expected between South Riverdale and other urban children. However, levels of lead in dust fall and house dust, and possibly paint, may account for the observed differences in blood lead levels.

In 1987, the MOEE established a Lead in Soil Committee to recommend a scientifically defensible soil removal guideline for lead-contaminated soil (MOEE, 1987). The Committee concluded that a quantitative assessment could not be made of the influence of individual environmental sources or pathways on blood lead levels of Ontario children due to limitations in the available data. In particular, no data were available on lead levels in house dust and paint. However, the Committee did conclude that levels of lead in air, soil, and house dust probably all contributed to the elevated blood lead levels in South Riverdale children. House dust lead was probably of critical importance.

The 1986 and 1987 Toronto Board of Health surveys in South Riverdale indicated that a significant decrease in blood lead levels had occurred since 1985 (Figure 4.1) (Macpherson, 1987; Langlois, 1992).

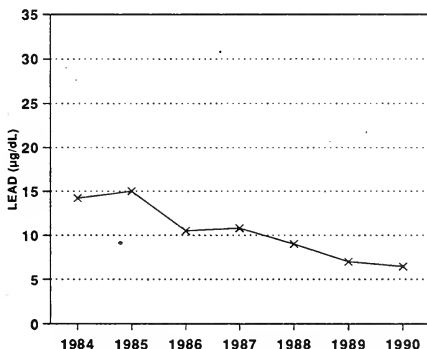


Figure 4.1 Mean Blood Lead with 95% Confidence Limits, Fall Screening (South Riverdale) Source: City of Toronto Board of Health Report

The reason for the decrease is unclear. Environmental levels of lead in soil and air did not change significantly during this period; and the soil removal program in South Riverdale was not initiated until the fall of 1987. It is possible that an effective community education program, emphasizing the potential for lead exposure through house dust and paint and the ways to minimize this risk, may have contributed to the observed decrease (Jenkins *et al.*, 1988). Subsequent to the lead abatement program in South Riverdale, blood lead levels have continued to decline (see Figure 4.2).

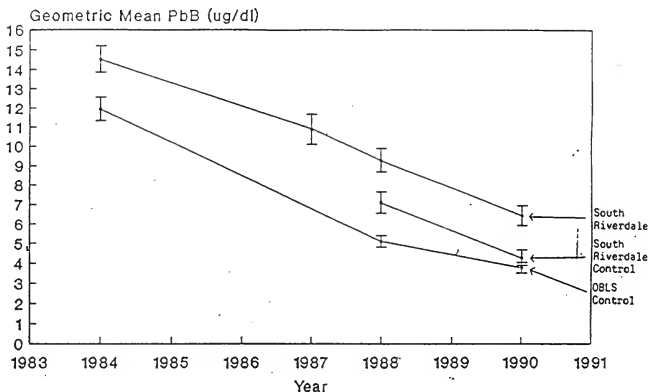


Figure 4.2 Average Blood Lead Levels: Lead Abatement Evaluation Project

4.2.1.3 The Northern Ontario Blood Lead Study (1987)

In 1987, the Ontario Ministries of Health and Environment investigated blood lead levels in children aged four to six years in seven Northern Ontario communities (Goss, Gilroy and Associates, 1989). The study provided data on blood lead levels of Northern Ontario children, and investigated the influence of numerous variables on PbB, including levels of lead in water, air and soil. A primary purpose of the study was to identify the demographic, environmental and lifestyle characteristics (risk factors) associated with higher blood lead levels. Concern had been expressed about the possible effects of lead in drinking water, because water in Northern Ontario may be more acidic and plumbosolvent than in the south.

In total, 835 children aged 3, 4, 5 and 6 years participated from two urban sites (Thunder Bay, North Bay); two town sites (Hearst, Sturgeon Falls); two rural sites (New Liskeard, Hymers area); and one remote site (Moosonee). The overall average blood lead level of the 835 children was 7.6 $\mu\text{g}/\text{dL}$ (geometric average, 7.0 $\mu\text{g}/\text{dL}$). With the omission of Moosonee, the overall geometric average blood lead level was 6.8 $\mu\text{g}/\text{dL}$. This level was much lower than the corresponding average found in the 1984 Ontario Blood Lead Study, and even lower when compared to other point source areas, such as Toronto South Riverdale. Only two children (2/835 or 0.2%) had PbB values above the alert level of 20.0 $\mu\text{g}/\text{dL}$; however, upon retesting, the children's PbB's were found to be normal. In the 1984 study, 54 children (4.2%) exceeded the alert level and the overall geometric average blood lead level was 10.3 $\mu\text{g}/\text{dL}$.

Boys had slightly higher blood lead levels than girls. However, there was no statistically significant difference in PbB between age groups in contrast to earlier studies. The analysis of PbB by site revealed the following differences: urban areas had the highest average, then towns, with rural areas the lowest. However, the remote area of Moosonee did not follow this pattern. The children's average PbB was comparable to that found in the urban areas surveyed in this study. The blood lead levels were well below the alert level of 20.0 µg/dL. Detailed analysis offered no apparent reasons for Moosonee's higher than expected values.

The blood lead levels were statistically significant when correlated with lead levels in air, soil, and residential drinking water. There was an association between blood lead levels and exposure to gasoline emissions as measured by proximity to gasoline stations and heavily travelled roads. This association disappeared when adjustment was made for air lead levels; the effect of gasoline emissions was directly associated with air lead levels. There was also an association between the socio-economic status of the parent and blood lead levels of children. The latter association persisted, even after adjusting for all environmental factors. There was no significant association between lifestyle factors and PbB, once the environmental and socio-demographic factors were taken into account.

Multi-variate statistical modelling identified the major risk factors that predicted blood lead level, as follows: level of lead in air, soil and drinking water, and the socio-economic status of parents. These factors explained 30% of the variance in PbB.

The variables associated with PbB included those previously measured in the 1984 study. In general, factors associated with gasoline consumption and industrial emissions were more prevalent in Southern Ontario and might explain the higher PbB. Alternatively, the overall variability in PbB in this study was smaller than in the 1984 study. In addition, there may have been a decreasing trend in lead concentrations in the environment over the period 1984-1987.

If the same risk factors are employed for prediction in this study as were used in the 1984 study, 25% of the variance in the PbB of the children in the 1987 Northern Ontario study could be explained. This figure of 25% represents a 4% improvement over that explained in the 1984 study. The influences of dietary lead, house-dust lead and paint lead were not assessed in this study.

4.2.1.4 The South Riverdale Lead Abatement Evaluation Study - Current Blood lead levels.

The South Riverdale Remedial Action Plan included the removal of lead-contaminated soil and house dust from residential sites in the vicinity of the secondary lead smelter (Section 4.2.1.2). The Hazardous Contaminants Branch of the Ministry of Environment and Energy and the Public Health Branch of the Ministry of Health, in association with the City of Toronto have sponsored a study which attempts to evaluate the influence of the remedial plan on community blood lead levels. Annual fall surveys of blood lead in the South Riverdale areas continue to be carried out. Blood lead levels of a control population in

Toronto in 1988 and 1990 were determined to provide blood lead data on urban children removed from an industrial point source. Control surveys of Toronto schools from the 1984 Ontario Blood Lead Study were also included for 1988, 1990 and 1992 to provide comparative time trend data. The study is nearing completion at the time of this report.

The blood lead distributions of the control groups provide valuable information on blood lead level trends in urban children in Southern Ontario. Although the data are limited in terms of the number of children and are restricted to the Toronto area, they do provide evidence that blood lead levels in urban children have declined significantly in recent years. In fact, the blood lead distribution in Ontario compares favourably with any other jurisdiction. The trends are illustrated in Figure 4.1. Geometric mean PbB's in the South Riverdale control groups were 7.1 $\mu\text{g/dL}$ ($N=157$) and 4.3 $\mu\text{g/dL}$ ($N=177$), in 1988 and 1990, respectively. In the 1984 Ontario Blood Lead Study control schools (children 4-6 years), geometric mean PbB's were 5.1 $\mu\text{g/dL}$ in 1988 ($N=256$) and 3.8 $\mu\text{g/dL}$ in 1990 ($n=245$). When compared to the mean levels of 11.9 $\mu\text{g/dL}$ ($N=11.9$) found in 1984, they represent an approximate 70% decline in the mean PbB of urban children. The fall 1992 survey of blood lead in the school-age children demonstrated an average blood lead of 3.5 $\mu\text{g/dL}$ (95% CI 3.1-3.8, $N=222$), with no significant change from the 1990 level (Smith, 1993). In 1990, approximately 3-4 % of children in the surveys exhibited blood lead levels of greater than 10 $\mu\text{g/dL}$.

4.2.2 Relationships between Environmental Exposure Levels and Blood Lead Levels

The exposure assessment in this criteria document focuses on strategies for predicting intake or uptake rates from multiple single media. Using modelling methods, the assessment provides a basis of predicting the relationship between these exposures and total daily intake or blood lead levels. Previous approaches have used epidemiological and human experimental data to derive mathematical relationships between environmental exposure levels (e.g., lead in drinking water) and increases in blood lead concentrations. This section provides a brief overview of the existing key information on relationships between lead levels in various media and human blood lead levels.

4.2.2.1 Relationships between PbB and Dietary/Drinking Water Lead

The relationship between PbB and lead in drinking water appears to be non-linear at water lead concentrations greater 100 $\mu\text{g/L}$ and varies considerably depending on the study. USEPA deduced a best estimate of 0.06 (units of ($\mu\text{g Pb/dL}$)/($\mu\text{g Pb/L water}$)) for the slope factor. The latter was associated with first draw water at lead concentrations below 100 $\mu\text{g/L}$ (USEPA, 1986; Pocock *et al.*, 1983). Lower slopes may be appropriate at higher water concentrations. At typical ambient drinking water levels, the relationship appears to be linear.

A more recent study by the Centers for Disease Control (CDC) was carried out in a Hawaiian population with a very low historical exposure to lead in air, soil, dust or paint (Maes *et al.*, 1989). The slope was estimated at 0.024 ($\mu\text{g Pb/dL}$)/($\mu\text{g Pb/L water}$) based on a multiple linear regression model. Fitting the data to a piecewise linear model yielded slopes factors of 0.02 at concentrations above 10 $\mu\text{g/L}$ and 0.13 below 10 $\mu\text{g/dL}$.

Dietary intakes of lead have been examined in a number of studies (Stuik, 1974; Cools *et al.*, 1976; Sherlock *et al.*, 1982; Ryu *et al.*, 1983; Lacey *et al.*, 1985). The derived relationships appear to be non-linear at dietary intakes greater than 200 $\mu\text{g Pb/day}$. USEPA has selected two studies to be the most appropriate for deriving dietary lead/blood lead relationships for adults: a duplicate diet study of 31 mothers and children in Ayr, Scotland (Sherlock *et al.*, 1982); and one in which 11 subjects were experimentally studied for blood lead response to oral doses of lead acetate (Cools *et al.*, 1976). The weighted mean of the studies is 0.032 $\mu\text{g/dL}$ increase in blood lead per $\mu\text{g/day}$ intake. This figure is five to six times lower than the comparable figure for infants at low exposure levels. At levels above 40 $\mu\text{g/day}$, a slope of 0.009 $\mu\text{g/day}$ is indicated (USEPA, 1988).

For infants, estimates of dietary lead/blood lead slopes have been made primarily on the basis of the Ryu infant dietary study (Ryu *et al.*, 1983). USEPA had calculated a slope of 0.16 $\mu\text{g/dL}$ per $\mu\text{g/day}$ based on these data, but now utilizes values of 0.2 to 0.25 based on a recent re-analysis (Marcus, 1989).

4.2.2.2 Relationships between PbB and Air Lead

A number of studies have provided data on blood lead in human populations and corresponding ambient air lead levels from which relational slopes have been calculated in children (Landrigan *et al.*, 1975; Roels *et al.*, 1976, 1980; Yankel *et al.*, 1977; Morse *et al.*, 1979; Angle and McIntire, 1979; Walter *et al.*, 1980). These slopes predict the increase in PbB per unit microgram per cubic metre ($\mu\text{g/m}^3$) increase in air concentration. Two types of slope factor have been derived: aggregate and disaggregate. Aggregate slope factors reflect the combined effect of air lead uptake through direct inhalation, as well as oral uptake of atmospheric lead deposited on soil, dust, crops and water. Disaggregate slope factors show the relationship between PbB and inhaled lead alone and are derived from cross-sectional studies where sufficient information is available on non-inhalation sources.

A critical evaluation by USEPA has provided aggregate slope factors, ranging from 2 to 20 ($\mu\text{g/dL}$)/($\mu\text{g/m}^3$) in young children with moderate levels of exposure (USEPA, 1988). Brunekreff has reported typical aggregate slope values ranging from 3-5 $\mu\text{g/dL}$ (Brunekreff, 1984). Results of another study suggest that the indirect contribution to increases in PbB is 4 to 5 $\mu\text{g/dL}$ above the direct inhalation contribution (Angle *et al.*, 1984).

Estimated disaggregate slopes have been derived from three studies of air lead/blood lead relationships in children (USEPA, 1986). The slopes were as follows: 1.92 ± 0.60 (Angle and McIntire, 1979), 2.46 ± 0.58 (Roels *et al.*, 1980) and 1.53 ± 0.84 (Walter *et al.*, 1980). The

median of these slopes is $1.97 (\mu\text{g/dL})/(\mu\text{g/m}^3)$. At high air lead concentrations, the relationship is non-linear. At air lead levels less than $3.2 \mu\text{g/m}^3$, there is no statistically significant difference between curvilinear and linear blood lead relationships.

Experimental studies in adults have measured changes in PbB in subjects exposed to lead aerosols (Kehoe *et al.*, 1961; Griffen *et al.*, 1975; Gross, 1979; Rabinowitz *et al.*, 1976, 1977; Chamberlain *et al.*, 1978). The weighted average slope in adult males was $1.64 \pm 0.22 (\mu\text{g/dL})/(\mu\text{g/m}^3)$ for all studies.

The results of the Isotopic Lead Experiment in Turin, Italy, showed that 25% of the blood lead in adults was derived from gasoline lead (Facchetti and Geiss, 1985). It should be noted that relatively high gasoline lead levels (0.6 g/L) and local conditions contributed to very high air lead levels ($1.7 \mu\text{g/m}^3$) at the time of the study. The study also showed that air lead levels directly affect street dust lead levels. The blood lead/air lead relationship observed in adults was $3.3 \mu\text{g/dL}$ per $\mu\text{g/m}^3$ of lead.

4.2.2.3 Relationships between PbB and Soil and Dust Lead

Some studies have found positive correlations between soil lead and PbB in children, particularly where soil lead levels exceed 1000 ppm. According to the Centers of Disease Control, concentrations of lead in soil or dust greater than 500 to 1000 $\mu\text{g/g}$ result in blood lead levels in children that exceed the background level (CDC, 1985). The range of reported average slope factors, which attempt to describe the relationship numerically, is 0.6 to 6.8 $\mu\text{g/dL}$ per 1000 $\mu\text{g/g}$ soil lead (USEPA, 1986a, based on Angle and McIntire, 1979 and Yankel *et al.*, 1977). It has been suggested that the value of 2 $\mu\text{g/dL}$ per 1000 $\mu\text{g/g}$ (Stark *et al.*, 1982) represents a reasonable median estimate (ATSDR, 1993).

As discussed in the 1987 MOEE Lead in Soil Committee Report (MOEE, 1987), studies relating soil lead to PbB are difficult to compare. The relationship depends on numerous factors, such as depth of soil lead, sampling methods, age of children, mouthing activities, cleanliness of the home and so forth.

A study in Derbyshire, England, concluded that soil lead contributed $0.6 (\mu\text{g/dL})/(\text{mg Pb/g})$ soil in a rural area where industrial point sources of lead no longer operate (Baltrop *et al.*, 1975). Lead-contaminated soil was covered by vegetation, which may have made soil lead less accessible as dust to children. Another study, however, demonstrated no apparent elevation in mean PbB (compared to low exposure groups) for children in two English villages with mean soil lead levels greater than 1000 $\mu\text{g/g}$ (Strehlow and Baltrop, 1988).

The soil lead/blood lead slope factor varies with the source of lead. In a more recent review of blood lead studies in mining areas, with mine waste but no recent history of smelting, blood leads were generally not elevated despite some very high soil lead concentrations (Steele *et al.*, 1990). Average PbB's were lower than expected when compared to studies of

urban communities or communities with operating smelters. The estimated average slope for mining sites is 1.7 $\mu\text{g}/\text{dL}$ per mg Pb/g soil.

The relationship between house dust lead levels and PbB has been well documented (USEPA, 1986). The contribution of house dust to the elevated PbB of children (aged 15 to 72 months) was examined in Baltimore. The children had been found to have PbB between 30 and 49 $\mu\text{g}/\text{dL}$. The study indicated that ingestion of lead-contaminated house dust was an important factor in elevating children's PbB and removal of lead-contaminated house dust could significantly lower it. The range of blood lead/house dust slope factors was 0.2 to 7.2 $\mu\text{g}/\text{dL}$ per mg Pb/g of dust (Charney *et al.*, 1983).

The effect of both soil and house dust lead levels on PbB has been demonstrated in several studies (Bornschein *et al.*, 1986; Rabinowitz *et al.*, 1985; USEPA, 1986 for summary). In the prospective study conducted in Cincinnati, 38% of the variation in PbB of children 18 months of age was accounted for by hand and dust lead. An indirectly mediated increase of 6.2 $\mu\text{g}/\text{dL}$ in PbB per 1000 ppm increase in soil lead was observed. Exterior environmental lead, such as soil lead, contributed to blood lead through the pathway soil lead to dust lead to hand lead to blood lead (Bornschein *et al.*, 1986).

As a result of the complexity of exposures to lead, determining the specific contribution of any particular environmental variable like soil or dust to PbB is extremely difficult. This is further confounded by other significant factors, such as socio-economic status and dietary exposure. For instance, the numerous variables studied in two Ontario blood lead studies (Duncan *et al.* 1985; GGA, 1988) were unable to account for more than 30% of the variations seen in children's PbB. The large number of derivations for the relationship between soil lead and PbB in different studies further reflects the difficulties in determining such associations.

4.2.3 Limitations of Epidemiological Blood Lead Studies

Many studies have attempted to estimate the contribution and significance of various sources of lead to blood lead levels. Unfortunately, comparison between these studies is difficult for several reasons: variations in the age of the population studied; the lack of standardized methods; and the statistical limitations inherent in each study. Site-specific factors, such as climate; the type of vegetation cover; and socio-economic status play a significant role in determining the relative influence of each source of lead. Another major confounding factor is that significant sources of lead exposure, e.g. dietary lead or lead in paint, are not part of these studies. As a result, much of the observed variance in blood lead levels cannot be explained through the variation in the environmental lead levels.

The multiple, interactive environmental pathways of lead exposure make it difficult to assess the specific contribution of any individual pathway to blood lead levels. Epidemiological

studies are complex and costly and, to date, no single epidemiological study has assessed the specific contribution of each environmental variable.

Regression analysis of the data generated in these studies, may be used to identify influential variables, but the analyses are limited in their capacity to assess the magnitude of the effect of each variable. For example, where elevated air lead levels contribute to elevated soil lead levels, and both air lead and soil lead contribute to house dust lead levels, regression analysis may indicate a significant relationship between blood lead and air lead, blood lead and soil lead, and blood lead and house dust lead. However, the analysis is limited in its ability to quantify the effect of a change in the level of lead in one environmental medium (e.g. soil) on blood lead levels.

A significant proportion of total lead exposure is associated with dietary factors and consumer products. Socio-economic status and occupational exposure may also play significant roles. This makes determination of the influence of the various environmental exposure pathways extremely difficult. For example, in the 1984 Ontario, Northern Ontario and South Riverdale studies, the variables studied were able to account for no more than 30% of the observed variation in blood lead levels.

4.3 ESTIMATES OF MEDIUM-SPECIFIC EXPOSURES AND TOTAL INTEGRATED EXPOSURE PROFILES

4.3.1 Multimedia Modelling Considerations

The exposure assessment exercise describes, in both qualitative and quantitative terms, the interaction of an environmental contaminant with the human population. For the purpose of assessing risk, this information may be used to estimate the dose encountered in the environment. The calculated dose can then be used to derive a dose-response relationship. In the case of lead, one must attempt to link sensitive populations, such as young children and the fetus, to concentrations of the chemical within the various routes of exposure. From these linkages, the contribution of individual pathways to the total daily intake can be estimated. The integration of these estimates from multiple exposure routes forms an overall picture of the probable exposures to lead.

Lead is a multimedia contaminant with human exposure occurring through environmental pathways (air, water, soil and dust); ingestion of food and water; and use of lead-containing consumer products. The complexity of the interactions between the main routes of exposure is illustrated in Figure 4.3. Airborne lead is the ultimate source of the major portion of environmental lead contamination. However, it is the lead deposited from air onto soils and dusts, which are then subsequently ingested, that is the primary intake factor for young children (Charney *et al.*, 1983; Bellinger *et al.*, 1986; Bornschein *et al.*, 1986). Children are particularly at risk of ingesting soil/dust lead as a result of normal hand to mouth activity and immature dietary habits, such as eating foods from dirty surfaces (Lin-Fu, 1972; Charney

et al., 1983). Such intake is of particular concern in areas with high atmospheric lead concentrations, as would be found near a point source. The ways in which a child might be exposed to lead in a household setting are depicted in Figure 4.4.

Constructing exposure estimates from different routes requires the following:

- consideration of relevant data on the ambient concentrations of lead in each media;
- data on consumption of and exposure to these media;
- data on trends in exposure levels over time; and
- data on uptake and absorption following exposure.

The available data are seldom comprehensive. Generally, assumptions must be made in the estimates of environmental concentrations, rates of intake and absorption factors. Thus, the exposure assessment presented is a model which predicts hypothetical doses not necessarily encountered. Rough estimates of the exposures of "typical" individuals living in Ontario are generated, recognizing that the exposure of a given individual may vary widely from these estimates because of factors such as age, activity, and geographical location. These media-specific estimates are deterministic in nature; that is, they are point estimates of average exposures that would be more precisely described by ranges. This type of modelling is deterministic in nature and does not provide a probability distribution. However, it is felt that the models developed are reasonably representative of the central tendency of exposure and can provide reasonable guidance as to the relative contribution of various sources and pathways to exposure. The assumptions regarding body weight and consumption of soil, water, breathing rates and so forth are summarized in Appendix F. Additional discussion of the assumptions underlying these rates, the concentrations selected or modelled, and analysis of special subpopulation scenarios of exposure is provided in the following sections.

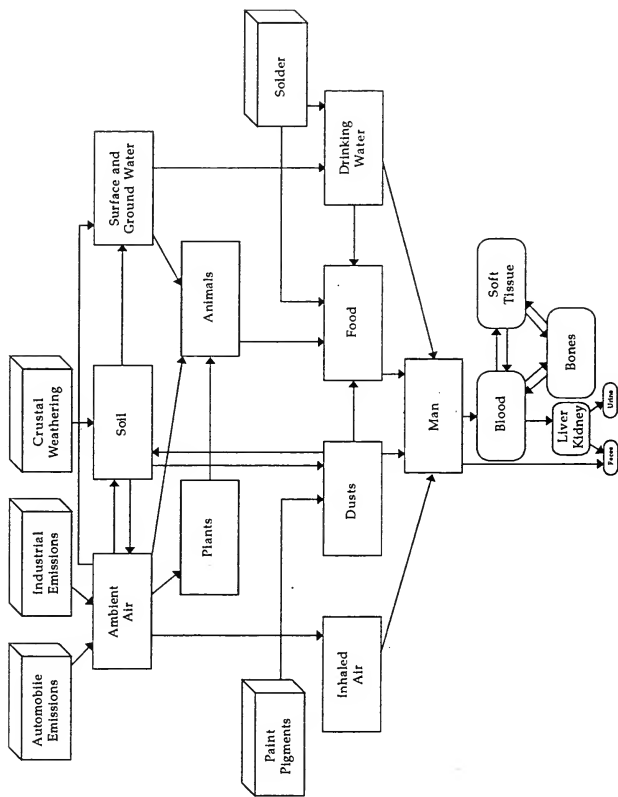


Figure 4.3

Pathways of lead from the environment to man, main compartments involved in partitioning of internal body burden of absorbed/retained lead, and main routes of lead excretion

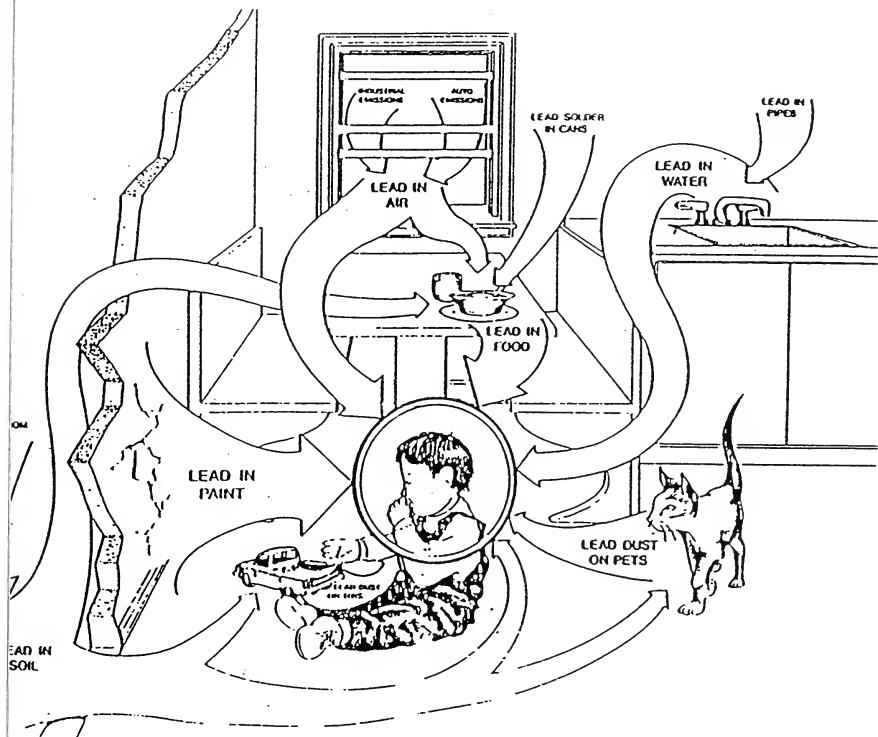


Figure 4.4 Ways in which children can be exposed to lead in the household

4.3.2 Lead in Food

The primary source of individual lead exposure is probably lead in food and beverages. The relative contribution to the integrated lead uptake will be elevated for populations without substantial exposure to other lead sources. Previously, Health and Welfare Canada estimated that 83% of a rural Canadian adult's total lead uptake was from diet, 60% for urban adults. For Canadian children one to three years old, food is thought to account for 41% of uptake for urban children, 73% for rural. The lower percentage contribution in urban populations is due to the larger contributions from atmospheric emissions by smelters and automobile exhaust. On an individual basis, the relative contribution of dietary lead is a direct function of age, body size, physiological state and type of diet.

To predict the quantity of lead ingested by Ontario adults and children through food, two pieces of information are needed. First, the food consumption rates and patterns of the populations of interest must be estimated as reliably as possible. The consumption rates are then combined with data on lead levels in specific foods to yield estimates of lead intake. The summation of the results from each food type provides a total estimated dietary intake.

4.3.2.1 Food Consumption Data

The best consumption figures for assessing dietary exposure are those of the 1970-72 Nutrition Canada Survey (NCS), which involved detailed dietary survey of over 13,000 individuals. Ontario information was a subset of the Canadian statistics. The NCS data were based on 24-hour recall by participants and provide age-specific consumption patterns. The nine major food classifications used were based on food groups developed by the United States Department of Agriculture. The major factor affecting the use of these data is the likely shift in food consumption patterns since the early 1970's, as population characteristics and available food types changed.

The diet of infants (under one year) is primarily milk-based and thus is considerably different from that of older children. Infants are therefore considered separately. However, the NCS data on infants was used in the analysis on infant dietary lead exposure (Dabeka and Mackenzie, 1987.) The NCS publication contains specific information on infant consumption.

4.3.2.2 Concentration of Lead in Food

Lead may enter food prior to human consumption, during production, harvesting, distribution, storage and cooking (USEPA, 1986). Potential means of lead contamination may include the following:

Production -

Lead may be present in the atmospheric fallout of airborne particulates onto crops, soils and forage areas. There is also a limited uptake of naturally occurring soil lead by plants through root systems.

Processing and packaging-

Food may come into contact with lead in the atmosphere, dusts, machinery, process water and most significantly, the lead-soldered seams of cans.

Preparation -

Lead may be present in water used for cooking or beverage preparation.

Serving or storage -

Food may come into contact with lead-containing pottery or utensils.

Many other sources of lead contamination probably remain to be determined.

Recent American studies suggest that lead concentrations in various foods may increase 2 to 12 times over background levels during processing and packaging (Wolnick *et al.*, 1983; USEPA, 1986). An important source of contamination is lead leached from lead-soldered cans. Lead-soldered cans, used for milk and formulae, appear to be the major contributor to infants total dietary lead intake (Dabeka, 1986). In the United States, the percentage of lead-soldered cans has declined from over 90% in 1979 to about 20% in 1986 (ATSDR, 1988). During this period, lead concentrations in canned foods decreased by 77% (NFPA, 1986) and lead in infant foods declined considerably (Jellinek, 1982). The decline was largely the result of regulatory activity.

Similar progress has been made in Canada. The goal of the voluntary phase-out program was to convert to alternative canning technologies by the mid-1980's (apart from a very few specialty cans). As of 1988, approximately 65% of lead-seamed cans had been converted (B. Huston, 1990). In some cases, such as beverage cans, conversion approached 100%.

Other changes which have reduced the lead content of canned foods are the introduction of low-lead solders; better industrial housekeeping procedures; and an improved soldering process, which reduces the splattering of solder inside cans. In addition, more foreign

countries are no longer using lead-soldered cans. Routine monitoring of foods, both domestic and imported, is carried out by the Federal Government.

Comprehensive information is not available on the lead concentrations in the Ontario food basket. However, some data exist for meats, sport fish, vegetables and alcohol (Appendix C). From 1980-83, the Ontario Ministry of Agriculture and Food (OMAF) carried out an elemental analysis on samples of kidney, liver and muscle from slaughtered Ontario beef, pork and poultry (Frank *et al.*, 1985). In general, the lead levels in organ meat were higher in chicken and pork than in beef and substantially higher in kidney/liver than in muscle tissue.

Data are available on the concentrations of lead in Ontario freshwater sport fish near certain sources of lead emissions. The average concentration in fish from the Blue Church Bay location in 1985 ranged from 0.10 to 0.88 µg/g. It is recognized, however, that this represents a worst case.

Lead concentrations have been analyzed in a variety of vegetables grown in the Holland Marsh area. Dry weight concentrations were highest in cauliflower at 40.2 µg/g (Czuba and Hutchinson, 1980).

The lead content of alcoholic beverages is routinely monitored by the Liquor Control Board of Ontario. In 1988, analyses of wine, beer and spirits/liquors showed mean levels (ng/g) of 9.2 (range 1.0-588.3), 2.5 (1.0-41.4) and 13.6 (1.0-1167.0), respectively (A. Karumanchiri, LCBO, personal communication, 1988). As for other food groups, wide variations were observed in the levels measured.

Detailed information available on lead concentrations in specific food groups, is provided by the Duplicate Diet Study of Canadian adults (Dabeka *et al.*, 1986) and an analysis of lead levels in 131 infant foods (Dabeka and McKenzie, 1987). In the 1981 survey, 24 hour duplicate diets, which included drinking water and snacks, were collected from 24 adults living in five Canadian cities, including Toronto. The mean and median concentrations and the range of mean lead concentrations are contained in Table 4.1. The overall range of lead levels was 8.8 ng/g to 654 ng/g, with a mean of approximately 32 ng/g.

Large variations in lead levels were found both between and within food categories. The levels, however, are in relatively good agreement with the findings of the more extensive adult Total Diet Study conducted by the United States Food and Drug Administration (USFDA) (Gartrell *et al.*, 1985). The highest level of lead found in a single food category was 6775 ng/g in a sample of parmesan cheese. A subsequent survey of 11 samples of parmesan cheese yielded a range of levels from 22 ng/g to 610 ng/g, with a mean of 101 ng/g. This agreed with the data on other cheeses.

TABLE 4.1 LEAD CONCENTRATIONS IN FOOD GROUPS IN CANADA, 1981 (ng/g)

FOOD CATEGORY	MEAN	MEDIAN	RANGE	TOTAL LEAD INTAKE (%)
Cereals	33.7	32.4	12-78	15.3
Water (consumed directly)	8.8	2.0	0.25-71	4.1
Coffee, tea, beer, soft drinks	10.9	8.8	0.05-29	15.4
Fruit juices, fruits	17.9	7.9	1.5-109	9.6
Dairy products, ice cream, eggs	8.9	3.3	1.2-82	4.5
Starch vegetables, rice, potato	21.8	16.9	5.5-84	6.4
Other vegetables, tomato products	49.3	31.7	0.62-254	-
Meat, fish, poultry	42.6	31.3	11-121	16.6
Misc. pie, chocolates, candy	117.6	33.1	14-1381	7.6
Cheese (other than cottage)	653.9	33.8	28-6775	3.5

Source: Dabeka *et al.*, 1987

The most recent and comprehensive data on lead levels in food in the Canadian diet are from a total diet study of lead and cadmium carried out in 1985 (Dabeka and McKenzie, 1991). The study analyzed canned and raw foods purchased at the retail level in six Canadian cities, including Ottawa, Ontario. The foods were prepared as would be done at home and then combined into 112 composites prior to analysis. The mean, median and range of lead concentrations were 29 ng/g, 14.7 ng/g and 1.42 to 407 ng/g. The mean level agreed well with the mean level of 32 ng/g found in the 1981 Canadian 24-hour duplicate diet survey. Analysis of components of the composites, as well as the composites themselves, revealed that some of the canned foods (luncheon meats, fish, beans, citrus fruits, apple sauce and cherries) contained appreciably higher lead levels than their fresh or frozen counterparts. Where lead-soldered cans were identified, the lead level only exceeded 100 ng/g in canned syrup, citrus fruits, tomato juice and fish. These data are judged to be the most appropriate for estimating dietary intake estimates and are reproduced in Table 4.2.

Data on the levels of lead in infant foods are contained in studies conducted by the same investigators (Dabeka and Mackenzie, 1987a, 1987b). The lead levels used to estimate the lead intake for infants are reproduced in Appendix H, including levels of lead in specific fruits, juices, or desserts. The mean levels of lead found in milk and infant formula ranged in concentration from 1.04 ng/g in human milk to 46.2 ng/g in canned ready-to-use formula (lead-soldered seams). A smaller sample of ready-to-use formula in lead-free cans had greatly reduced levels of 1.7 ng/g.

TABLE 4.2 LEAD EXPOSURE ASSESSMENT-GENERAL DIETARY INTAKE

FOOD COMPOSITE	LEAD LEVEL (ng/g)	FOOD CONSUMPTION (g/person/day)					ESTIMATED LEAD INTAKE (ug/day)					
		0-6 mo. m & f	.5-4 yr. m & f	6-11 yr m & f	19 yr. m & f	20+ yr. m & f	0-6 mo. m & f	.5-4 yr. m & f	5-11 yr m & f	19 yr. m & f	20+ yr. m & f	
MILK AND DAIRY PRODUCTS												
1. Milk, whole	2.5	274.16	377.08	323.16	255.65	138.24		0.69	0.94	0.81	0.64	0.35
2. Milk, 2%	1.9	188.6	194.5	185.61	194.75	60.64		0.36	0.37	0.35	0.37	0.12
3. Milk, skim	3.3	21.2	59.67	55.57	72.65	30.83		0.07	0.20	0.18	0.24	0.10
4. Instant breakfast prep.	4.7											
5. Cream	2.7	0	1.63	2.83	2.65	10.19		0.00	0.00	0.01	0.01	0.03
6. Ice Cream	6.2	1.37	15.35	25.69	25.78	12.8		0.01	0.08	0.13	0.13	0.07
Ice Cream, Chocolate	9.9											
Yogurt, Mixed	4.8	0	0.78	0.48	0.87	1.54		0.00	0.00	0.00	0.00	0.01
Yogurt, Plain	3.4											
Cheese	16.2	0.11	2.66	3.16	5.66	8.33		0.00	0.04	0.05	0.09	0.13
Cheese, Cottage	6.5	0	1.73	1.33	1.74	5.35		0.00	0.01	0.01	0.01	0.03
Cheese, processed	17.6	0.08	3.59	4.92	6.43	3.81		0.00	0.06	0.09	0.11	0.07
Butter	6.5	0.73	7.06	12.94	16.67	13.61		0.00	0.05	0.08	0.11	0.09
MEAT AND POULTRY												
Beef Steak, Cooked	10.2	0.07	3.09	7.37	10.89	17.39		0.00	0.03	0.08	0.11	0.18
Beef Steak, Raw	16.2											
Roast Beef	16.6	0.27	6.49	12.21	23.27	27		0.00	0.11	0.20	0.39	0.45
Ground beef, Cooked	12.4	31.38	20.05	19.23	30.84	21.61		0.39	0.25	0.24	0.38	0.27
Ground Beef, Raw	7.8											
Pork, Cooked	6.3	0	7.24	11.88	22.74	22.73		0.00	0.04	0.06	0.12	0.12

Rolls and Biscuits	14.5	0	3.64	11.63	15.92	10	0.00	0.05	0.17	0.23	0.15
Rolls and buns only	13.9										
Wheat Flour	1.6	0.28	3.86	10.38	5.17	6.93	0.00	0.01	0.02	0.01	0.01
Cake and Muffins with Raisins	18.9										
Cake and Muffins	28.2	0.19	8.59	25.62	42.52	20.37	0.00	0.23	0.67	1.11	0.53
Cookies, all	16.3	1.5	18.87	26	23.08	15.58	0.02	0.31	0.42	0.38	0.25
Cookies, oatmeal, arrowroot	14.4										
Cookies, chocolate chip	21.7										
Danish and Donuts	18.9	0	3.8	5.39	9.53	5.49	0.00	0.07	0.10	0.18	0.10
Crackers	23.1	0.04	4.83	5.14	5.67	3.45	0.00	0.11	0.12	0.13	0.08
Waffles and pancakes	21.1	0	2.16	2.93	3.37	2.04	0.00	0.05	0.06	0.07	0.04
Cooked wheat cereal	1.7	13.5	13.94	5.72	4.73	6.53	0.02	0.02	0.01	0.01	0.01
Cream of Wheat, dry	8.6										
oatmeal cereal	6.7	33.12	20.86	19.95	12.26	16.44	0.22	0.14	0.13	0.08	0.11
oatmeal cereal, dry	3.1										
Corn cereal	6.4	1.07	3.42	5.37	3.4	1.82	0.01	0.02	0.03	0.02	0.01
Wheat and bran cereals	20	0.09	3.37	3.37	3.35	2.31	0.00	0.07	0.07	0.07	0.05
Rice cereal, cooked	9.5	0	6.73	13.98	14.58	15.14	0.00	0.06	0.13	0.14	0.14
Rice cereal, dry	5.3										
Apple pie	13.1	0	2.02	3.87	3.71	9.25	0.00	0.03	0.05	0.05	0.12
Pies, other, mix	37.3	0.08	3.68	10.35	10.77	11.7	0.00	0.14	0.39	0.40	0.44
Pie, no raisins	19.8										
Raisin pie	247.4										
Pizza	18.9	0	0.12	3.09	6.09	1.74	0.00	0.00	0.06	0.10	0.03
Pasta, canned	7.9	0	17.67	36.9	46.99	16.81	0.00	0.14	0.29	0.37	0.12
pasta, plain, cooked	14.1	0	10.85	28.24	10.32	13.47	0.00	0.15	0.37	0.15	0.19
VEGETABLES											
Corn, raw and canned, cooked	51.8	0.56	9.9	17.6	12.02	6.16	0.03	0.51	0.91	0.62	0.42
Corn, raw	181.5										
Corn, kernal, canned	5.7										
Potatoes, raw	7.8	0	0.25	0	0	0.04	0.00	0.00	0.00	0.00	0.00
Potatoes, baked	40.6	0	2.08	2.95	3.05	4.92	0.00	0.08	0.12	0.12	0.20

Potatoes, boiled, skins	4	0	2.13	1.91	3.54	5.43	0.00	0.01	0.01	0.01	0.02
potatoes, boiled, peeled	3.8	7.51	45.22	77.66	100.98	82.11	0.03	0.17	0.30	0.30	0.31
French fries	5.1	0.01	18.48	22.78	33.02	20.68	0.00	0.09	0.12	0.17	0.11
Potatoe chips	12	0	1.64	6.18	7.91	1.31	0.00	0.02	0.06	0.09	0.02
Cabbage, cooked and coleslaw	4.8	0	2.89	5.05	8.21	10.26	0.00	0.01	0.02	0.03	0.05
Celery	44.9	0.03	1.59	2.43	3.45	8.34	0.00	0.07	0.11	0.15	0.37
Peppers, green and red	9.7	0	0.05	0.27	0.43	1.28	0.00	0.00	0.00	0.00	0.01
Lettuce	2.3	0	2.37	4.49	8.21	12.7	0.00	0.01	0.01	0.02	0.03
Cauliflower, raw and cooked	63.7	0	0.28	0.11	1.11	1.48	0.00	0.02	0.01	0.07	0.09
Cauliflower, raw	1.9										
Broccoli, raw and cooked	18	0.87	0.34	1.34	0.2	2.19	0.01	0.01	0.02	0.00	0.04
Beans, raw and canned, cooked	72.6	0.32	2.68	4.27	4.49	8.82	0.02	0.19	0.31	0.33	0.50
Beans, raw	13.3										
Beans, canned	120.2										
Peas, raw and canned, cooked	14.7	31.1	6.1	8.09	7.66	9.34	0.46	0.09	0.09	0.11	0.14
Peas, raw	9.6										
Peas, canned	23.9										
Carrots, raw and canned, cooked	9.7	1.39	8.14	10.34	11.08	14.19	0.01	0.08	0.10	0.11	0.14
Carrots, raw and canned											
Carrots, raw	9.1										
Carrots, canned											
Onions, cooked and raw	8.1	0	0.89	2.45	3.05	6.15	0.00	0.01	0.02	0.02	0.05
Onions, raw	9.4										
Turnips, parsnips	15.8	0.69	2.4	3.51	3.29	5.69	0.01	0.04	0.05	0.05	0.09
Tomatoes, raw and cooked	2.5	0	3.19	7.47	11.16	17.9	0.00	0.01	0.02	0.03	0.04
Tomatoes, cooked	1.4										
Tomatoes, raw	2										
Tomato juice, canned	24	0	5.28	4.52	5.64	10.02	0.00	0.13	0.11	0.14	0.24
Tomatoes, canned	109.4										
Mushrooms, raw and canned, cooked	44.6										
Mushrooms, raw	70.9	0	0.47	0.86	2.11	1.63	0.00	0.03	0.06	0.15	0.12
Mushrooms, canned	82.9										
Mushrooms, raw	8.3	0	3.47	8.27	11.27	11.37	0.00	0.03	0.07	0.09	0.09
Cucumber, raw, pickled											
Cucumber, raw	8.1										

FRUIT AND FRUIT JUICES

Citrus fruits, raw	18.3	0	11.47	24.7	22.29	33.25	0.00	0.21	0.45	0.41	0.61
Citrus fruits, canned	407.4	0	0	0.17	0.04	0.18	0.00	0.00	0.07	0.02	0.07
Citrus juice	19.7	3.48	34.61	22.54	32.98	35.01	0.07	0.68	0.44	0.65	0.69
Citrus juice, canned	17.8	11.82	9.69	12.98	11.05	13.38	0.21	0.17	0.23	0.20	0.24
Apples	18.2	1.15	26.78	41.38	33.85	20.52	0.02	0.49	0.75	0.62	0.37
Apple juice, canned and bottled	24.9										
Apple juice, bottled	10.4										
Apple juice, canned	23.3	14.98	44.21	26.86	9.65	13.3	0.35	1.03	0.62	0.22	0.31
Apple sauce, canned and bottled	48.2										
Apple sauce, bottled	12.7										
Apple sauce, canned	102.3	1.45	3.91	8.81	3.16	5.97	0.15	0.40	0.90	0.32	0.61
Bananas	1.9	3.25	12.98	21.42	11.19	12.82	0.01	0.02	0.04	0.02	0.02
Grapes		0	0.82	1.62	2.67	2.94	0.00	0.00	0.00	0.00	0.00
Grape juice, bottled	21.5	0	6.27	2.52	5.02	2.15	0.00	0.11	0.05	0.11	0.05
peaches, canned and raw	119.7	0.6	12.25	10.27	6.58	10.17	0.06	1.47	1.23	0.79	1.22
peaches, raw											
Pears, raw and canned	97.6	73.53	18.1	6.7	4.06	7.73	7.18	1.77	0.85	0.40	0.75
Pears, canned											
Pears, raw	8.6										
Plums, prunes, dried and canned	206.7	0.85	2.15	2.72	2.64	4.74	0.20	0.44	0.56	0.55	0.96
Cherries, raw and canned											
Cherries, canned	203.8										
Mellons	1.9	0	1.18	7.39	3.82	9.53	0.00	0.00	0.01	0.01	0.02
Strawberries	7.6	0	3.01	7.56	5.39	7.75	0.00	0.02	0.06	0.04	0.06
Blueberries	21.2	0.87	0.67	1	1.51	1.99	0.01	0.01	0.02	0.03	0.04
Pineapple, raw and canned	15.8	0	0.7	1.68	1.68	2.22	0.00	0.01	0.03	0.03	0.04
pineapple, fresh	15.2										
Pineapple, canned	31.3										

FATS AND OILS

Cooking fats and salad oils	3.1	0	1.23	2.21	3.97	4.95	0.00	0.00	0.01	0.01	0.02
Margarine	6	0.02	2.65	6.13	8.34	6.23	0.00	0.02	0.04	0.05	0.04
Peanut Butter	15.2	0.18	2.98	6.08	6.8	3.52	0.00	0.05	0.09	0.10	0.05

SUGAR AND CANDIES

Sugar	1.9	1.54	7.08	11.66	14.46	19.2	0.00	0.01	0.02	0.03	0.04
Syrup	69	3.13	2.89	6.45	6.59	4.94	0.22	0.20	0.45	0.39	0.34
Jams and Jellies	19.2	0.28	3.55	6.76	9.63	6.14	0.01	0.07	0.13	0.18	0.12
Honey	41.8	1.3	0.86	2.02	1.88	2.17	0.05	0.04	0.08	0.08	0.09
Pudding, canned, mixed, powder, pre	8.8	18.13	13.16	8.85	10.59	8.78	0.16	0.12	0.08	0.09	0.08
Pudding, chocolate powder and cann	30.3										
Candy, chocolate bars	40.2	0.18	3.14	5.45	8.1	3.58	0.01	0.13	0.22	0.33	0.14
Candy other	51.5	0.01	5.36	8.47	10.39	4.58	0.00	0.28	0.44	0.54	0.24

BEVERAGES

Coffee	5.2	0	6.48	11.69	83.95	347.77	0.00	0.03	0.06	0.44	1.81
Tea	9.2	0	8.47	22.2	81.64	354.13	0.00	0.08	0.20	0.75	3.26
Soft Drinks	5.1	2.39	100.33	193.57	240.7	109.91	0.01	0.51	0.99	1.23	0.56
Wine	140.6	0	0.02	0.73	1.84	23.54	0.00	0.00	0.10	0.26	3.31
Wine and beer, cans and bottles	78.9	0	1.22	1.93	21.44	121.25	0.00	0.09	0.15	1.65	9.32

TOTALS

	11.34	17.72	23.32	27.16	40.17	
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4.3.2.3 Estimated Daily Intakes of Lead in Food

4.3.2.3.1 Young Children and Adults

Dietary intake studies conducted in Canada in the early 1970's reported mean daily adult intakes to be 138 µg/day (Kirkpatrick and Coffin, 1974) and 106 µg/day (Meranger and Smith, 1972). The 1981 Duplicate Diet Study estimated mean dietary intakes for Canadian adults to be 53.8 µg/day or 0.80 µg/kg/day (Dabeka *et al.*, 1987). Calculated estimates of dietary lead intake for different Ontario age groups in the early to mid-1980's are outlined in Appendix G. The data are based on the duplicate diet survey data as applied to Ontario food consumption figures across large food groups. Dietary lead intake increases from 37.4 µg/day for children aged one to four years, to a maximum of 84.8 µg/day for adult males, aged 20 to 39 years. However, when the figures are adjusted for body weight, intakes for children under 11 years of age are at least 60% greater than for teenagers or adults. Adult women tend to have lower intakes than men, probably due to lower overall food consumption rates. These estimates agree well with those previously made by the Foods Directorate of Health and Welfare Canada in 1988: 37 µg/day (2.6 µg/kg/day) in a one to four year old child and between 46.1 and 84.8 µg/day (0.73 - 1.19 µg/kg/day) in adult groups.

Based upon trial investigations for the Canadian Total Diet Program conducted in 1985, the estimated dietary ingestion of lead by all segments of the population was 36.4 µg/day (Dabeka and McKenzie, 1991). This estimate is based upon one of two data sets from food collected in the Ottawa area. The data utilized was the set selected by the authors for their calculations. In order to estimate more recent daily intakes for Ontario subpopulations, published concentrations of lead in 100 food composites from this study and age-specific Canadian food consumption data are combined. The average intake figures are based on consumption calculations for Canadians, assuming that these figures also apply to Ontarians. The consumption figures for Canadians are preferred because they have been adjusted to reflect current consumption patterns and are consistent with representative diet figures. Sex-specific estimates were not made as consumption values provided are for males and females combined.

Estimated daily intakes of lead from food, based upon the summation of 112 food grouping intakes, are presented in Table 4.3. Highest total intake increases with age from 17.7 µg/day in young children to approximately 40 µg/day in adults. Infants and children are expected to have the highest exposure on a body weight basis at 1.6 and 1.4 µg/kg/day, respectively, compared to adult values of 0.6 µg/kg/day. It should be noted that an additional five Canadian cited have been surveyed since 1985 (1986-1988) and it would be expected that there would be same variation between different areas of the country.

A subgroup that requires additional consideration are women of child-bearing age. The current analysis does not allow for this specific calculation; however, previous estimates (Appendix G) indicate that women aged 20-39 years are likely to have significantly lower intakes than men. Therefore the adult value of 0.6 µg/kg/day is probably an overestimate for this group. In the last Nutrition Canada Survey (1972), pregnant women had a pattern of food consumption similar to that of 20 to 39 year-old females, except for modest increases in milk and fruit consumption. Estimated lead intakes would not differ significantly and would be unlikely to exceed 0.6 µg/kg/day on average.

TABLE 4.3 DAILY INTAKE OF LEAD FROM FOODS IN CANADIAN POPULATION BASED ON 1985 TOTAL DIET SURVEY

AGE GROUP	INTAKE OF LEAD ($\mu\text{g/kg/day}$)	BODY WEIGHT (kg)	INTAKE ($\mu\text{g/day}$)
0 - 6 months	1.7	7	11.3
7 mo.- 4 years	1.4	13	17.7
5 - 11 years	0.86	27	23.3
12 - 19 years	0.48	57	27.2
20 + years	0.57	70	40.2

4.3.2.3.2 Infants - Not Breast Fed

Previously reported estimates of the average dietary lead intakes by Canadian infants are $6.64 \mu\text{g/kg/day}$ (Kirkpatrick *et al.*, 1980) and $5 \mu\text{g/kg/day}$ (Nutrition Foundation's Expert Advisory Committee, 1982). Both are much higher than the current estimate of $1.6 \mu\text{g/kg/day}$. The principal reason may be the reduced use of lead in the Canadian canning industry and the lower limits of detection in the analytical methodology. Both are factors in the lower levels of lead reported in individual food categories (Dabeka, 1986; Dabeka and Mackenzie, 1987).

Dabeka examined lead in 282 infant formulas and evaporated milk (Dabeka, 1989). On an "as sold" basis, lead levels in ready-to-use, concentrated liquid, and powder formulas averaged 1.6, 3.7 and 12.6 ng/g , respectively. Dietary intakes of 0-12 month old children from food (and water used to dilute concentrated foods) were estimated at an average of $13.2 \mu\text{g/day}$ ($1.9 \mu\text{g/kg/day}$), a value which agrees well with the value estimated in Table 4.3. The estimated intake assumes that the infant is fed cow's milk. This lead intake falls close to the intake of concern of $1.85 \mu\text{g/kg/day}$ developed in the document but below the FAO/WHO provisional tolerable daily intake of $3.6 \mu\text{g/kg/day}$. For infants fed evaporated milk stored in lead-soldered cans intakes can be significantly higher (Section 4.4.4.).

4.3.2.3.3 Infants -Breast Fed

Recent interest has emerged in the toxicokinetics of lead during pregnancy and lactation, as mobilization of lead from the bone pool will increase during these periods. External exposures influence breast milk levels. Urban populations generally have higher levels than rural populations (Sternowsky and Wessolowski, 1985). The relationship between maternal PbB and lead in breast milk has not been well studied. Ong found a correlation ($r=0.29$), suggesting that transfer of lead from maternal tissues to breast milk is possible, but the metabolic mechanisms are complicated (Ong *et al.*, 1985). Breast milk concentrations may increase over lactation although no comprehensive studies have been done. Lead has also

been found in breast milk at concentrations higher than those found in plasma at the same time (Wolff, 1983).

It is now felt that the majority of infants are fed largely by breast feeding, therefore these estimates are considered to form the basis for the dietary exposure for the average infant. Approximately 80% of women initiate breast-feeding and 30% continue through 6 months (HWC, 1990). Dabeka and co-workers have also done a detailed analysis of lead in human milk in Canadians (Dabeka *et al.*, 1986). The mean, median and ranges of 210 samples were 1.04 ng/g, 0.55 ng/g and range 0.5 to 15.8 ng/g, respectively. Although no intake estimate is provided, assuming an average consumption of 750 mL of milk per day, the estimated mean intake is 0.78 µg/day or 0.11 µg/kg/day. Thus, the exposure from breast milk is small and likely less than that associated with formula or mixed diet. Infants fed formula from lead-soldered cans may encounter much higher exposure.

4.3.2.4 Trends Analysis of Dietary Exposures

Dietary lead is a large contributor to chronic daily exposure, so that careful consideration of trends is essential. A limitation with the following analysis is that the most current food survey was conducted in 1985. Food baskets collected between 1986 and 1988 in other Canadian cities have been analyzed but the results were not published at the time of this report. It is probable that lead levels have declined in Canadian foods since the 1980s; however, the trend has not been systematically monitored. Investigations of dietary lead sources in the United States may have bearing on this question. Estimated average consumption rates for adults in the United States include 60 µg/day (Mahaffey *et al.*, 1975), 95 µg/day (Podrebarac, 1984) and 82 µg/day (Gartrell *et al.*, 1986). Data from the recent Total Diet Study by the USFDA indicate 35-37% reductions in dietary lead intake for young children between 1982-84 and 1984-86 (ATSDR, 1988). Similarly, estimates made using the USEPA Multiple Source Food Model, suggest a decline in dietary lead intake for a two year old child from approximately 45-50 µg/day in 1978 to 13 µg/day in 1985 (Flegel *et al.*, 1988). The trend data in the Multiple Source Food Model allow the projection of dietary intake for American children for 1990-1996. Two-year olds are predicted to have an average intake of 10.4 µg/day in this period (Cohen, 1988). More recent estimates from the USFDA Total Diet Survey suggest even lower values of intake of 3.9 µg/day for a child in 1990.

Estimated food intakes for various time periods are provided in Table 4.4 for Ontario/Canadian individuals and are compared to USFDA diet figures. The unpublished USFDA figures were provided by one of the authors of the Total Diet Survey (E. Gunderson, personal communication, 1991). The USFDA data indicate a declining trend for lead in food, and therefore in dietary intake of lead. This is most notable for young children. Values levelled out in 1988 and 1990, possibly reflecting the virtual elimination of lead in domestic canning processes and of mobile sources of airborne lead. Similar trends are suggested by Canadian data, although a systematic determination of declines in specific foodstuffs has not been made (Figure 4.5). The Canadian values cited are not directly comparable because different consumption figures were used in deriving the values from the various studies.

TABLE 4.4 TRENDS IN DIETARY LEAD EXPOSURE ESTIMATES

YEAR	CANADA/ONTARIO		U.S. FOOD AND DRUG ADMINISTRATION ⁵	
	INTAKE µg/day (µg/kg/day)			
	CHILD	ADULT	CHILD	ADULT
1980			38.5	83
1982	37.4 (2.6) ¹	63.8 (0.9) ¹ 53.8 (0.8) ³	25.0	57
1984			19.9	38
1986	17.7 (1.2) ²	36.4 (0.6) ⁴ 40.1 (0.6) ²	12.5	23
1988			4.75	9
1990	6-8 (0.5-0.6) ⁶	26 (0.37) ⁶	3.9	8.1
1993	6 (0.5) ⁶	12 (0.17) ⁶		

Source: Data based on average Food Consumption Figures from Nutrition Canada Survey

¹ Foods Directorate, Health and Welfare Canada

² MOEE, 1993. This document- Based on total diet survey data.

³ Dabeka and MacKenzie, 1987. Duplicate diet survey.

⁴ Dabeka, 1991. Total Diet Survey.

⁵ Gunderson, 1991 (personal communication)

⁶ Projected based on estimates trends and U.S. figures.

The declining trend is attributed to the following three factors:

- reductions in ambient particulate fallout to crops as a result of phasing out leaded gasoline;
- the phase-out of lead-soldered cans by manufacturers; and
- a decline in lead levels in water used in food processing and preparation.

Comparison of the United States and Ontario estimates for children gives rise to interesting interpretational questions. Canadian estimates are somewhat higher than American figures. What may account for this difference and to what degree would a large difference be expected? The differences may in part be due to the following:

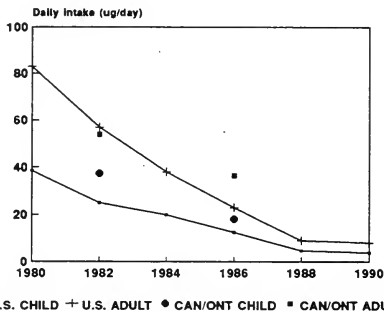


Figure 4.5 Estimated Food Intakes in U.S. and Canadian Populations: 1980 - 1990

- earlier phase-out of leaded gasoline in the United States
- greater frequency of American food basket surveys
- differences in study methods, particularly as the Canadian studies utilize more sensitive analytical techniques with lower detection limits
- differences in consumption patterns for specific food types, and
- differences in importation patterns of canned goods.

On the other hand, some similarity in intakes might be expected, particularly if comparing, for example, northeastern states to Ontario. Many foods may be obtained from common sources and reliance on locally grown foods is probably less significant than in the past.

The remaining question for estimating exposure for Ontario populations is what decline in lead levels and intake through foods would be expected between 1985 and the present. Intake estimates declined by roughly 50% between 1981 and 1985. Mean lead levels in food, however, do not appear to have declined in this period, according to the two studies by Dabeka and MacKenzie, although the upper end of the range is roughly one-third lower in the latter study. If one considers Canadian intake estimates for children to be roughly 50% higher than American values in a corresponding year, then an intake of 6 $\mu\text{g}/\text{day}$ would be suggested based on the 1990 U.S. figure of 3.9 $\mu\text{g}/\text{day}$, or less than 50% of the 1985 estimate. A projected estimate for 1990 assuming a continuation of the trend between 1981 and 1985

and assuming a further 50% reduction would be roughly 8 $\mu\text{g}/\text{day}$, thus the 1990 estimated range of 6-8 $\mu\text{g}/\text{day}$. Because of the voluntary elimination of lead in cans in the late 1980s, the lower end of the range is selected as the 1993 estimated for Ontario/Canadian children. No significant decline is expected between 1990 and the present and this is supported by observation of average blood lead levels in school age children in the Toronto area (see Section 4.2.1.4). The significant declines in observed children's blood lead levels in Ontario over the last decade would be congruent with the suggested trends presented here. Cognascent of the differences in U.S. and Canadian methods, the U.S. data are utilized here in a relative not absolute manner.

If adult intakes are roughly twice child intakes, then adult intakes may have declined to some point between approximately 12-16 $\mu\text{g}/\text{day}$ in 1990 and 12 $\mu\text{g}/\text{day}$ projected to 1993. Therefore estimates of the average dietary intakes for children and adults currently are likely quite lower than our estimates from the actual 1985 data may suggest. This hypothetical extrapolation to lower values is limited by the lack of current survey data on lead levels in food.

4.3.3 Estimation of Daily Intake of Lead from Ambient Air

Lead is present in air as a result of current industrial emissions, automotive exhaust and re-entrainment of lead-contaminated dusts. The primary contribution to air lead levels is the use of leaded gasolines. Fallout from industrial plants that process lead can produce more severe effects on a local scale. However, with the accelerated phase-out of lead in gasoline within Canada, emissions of concern will be almost exclusively related to confined localities surrounding significant point sources.

Routine monitoring of ambient air lead levels is carried out by the MOEE in locations throughout the province. The following observations were considered significant for modelling of exposure:

- The annual geometric mean for lead particulate levels decreased substantially from 0.4 $\mu\text{g}/\text{m}^3$ in 1978 to 0.1 $\mu\text{g}/\text{m}^3$ in 1987 (MOEE, 1988); annual geometric mean values for 1989 and 1990 are generally below 0.05 $\mu\text{g Pb}/\text{m}^3$ in urban locations and for most rural locations, the annual mean is 0.05 $\mu\text{g}/\text{m}^3$ or less (MOEE, 1991).
- Four monitoring stations exceeded the current Ontario ambient air quality guidelines of 5 $\mu\text{g}/\text{m}^3$ (24 hour) during 1987. All the stations were located within 100 metres of two secondary lead smelter operations in the Toronto area: the Canada Metal Company in South Riverdale and the Tonolli Company of Canada in Mississauga. The highest single value recorded was 38.7 $\mu\text{g}/\text{m}^3$ near the Canada Metal Company. The highest geometric mean value was 2.1 $\mu\text{g}/\text{m}^3$ at the Tonolli location. In 1991, the monthly geometric mean levels at stations 31058 (Linkbelt) and station 31065 (A.R. Clarke) near the Canada Metal Company ranged from 0.08 to 0.59 $\mu\text{g}/\text{m}^3$. The maximum measured daily value was 13.8 $\mu\text{g}/\text{m}^3$.

Hi-Vol sampling data collected by MOEE in Northern Ontario indicate that the mean airborne lead level during 1985-87 was $0.15 \mu\text{g}/\text{m}^3$. The latter is an arithmetic mean derived from pooled data from all regional Hi-Vol samples (GGA, 1988).

From this information, the lead concentration level selected for the exposure analysis was $0.05 \mu\text{g}/\text{m}^3$ for urban locations (non-point source) to reflect an "upper limit" of annual means for urban localities. Most cities lie in the 0.01 to $0.03 \mu\text{g}/\text{m}^3$ range. Rural areas would be expected to have even lower levels, because of lower traffic densities and lack of proximity to industrial lead emissions. A value of one-half this level was selected at $0.025 \mu\text{g}/\text{m}^3$.

The inhalation exposure analysis for various population subgroups living in urban areas (non-point source) is presented in Table 4.5. To account for differences in indoor and outdoor exposure, time-weighted average concentrations have been estimated, based on the indoor times indicated in the table. A range of indoor/outdoor air lead concentrations have been determined for different cities and building types, using ratios between 0.3 and 0.8 (USEPA, 1986). The present analysis uses a ratio of 0.5, the approximate midpoint of this range. The ratio will be affected by several parameters, including lead particle size; housing conditions; seasonal changes in home activities; and meteorological conditions, particularly wind and rainfall. Point sources involve larger, less mobile particles; hence, the ratio is estimated at 0.3 (Cohen and Cohen, 1980).

TABLE 4.5 ESTIMATED DAILY INTAKES OF LEAD VIA INHALATION FOR ONTARIO URBAN POPULATIONS

Population Subgroup	FACTOR					
	Air lead concentration ($\mu\text{g}/\text{m}^3$)	Indoor/outdoor concentration ratio	Time spent outdoors (hours per day)	Time weighted average concentration ($\mu\text{g}/\text{m}^3$) ¹	Volume inhaled (m^3/day)	Inhaled daily intake ($\mu\text{g}/\text{day}$) ($\mu\text{g}/\text{kg}/\text{day}$)
INFANTS (0-1 yr)	0.05	0.5	1.5	0.026	2.5	0.065 0.01
CHILDREN (1-4 yr)	0.05	0.5	3.0	0.028	5.0	0.14 0.01
CHILDREN (5-11 yr)	0.05	0.5	5.0	0.03	9.0	0.27 0.01
ADULTS (20+ yr)	0.05	0.5	5.0	0.03	22.0	0.66 0.01

¹ Sample calculation

$$\begin{aligned}
 \text{Time weighted average} &= (\text{air lead outdoor} \times \text{fraction of time outdoors}) + (\text{air lead outdoor} \times \text{I/O ratio} \\
 &\quad \times \text{fraction of time indoors}) \\
 &= (0.05 \mu\text{g}/\text{m}^3 \times 1.5 \text{ hr}/24\text{hr}) + (0.05 \mu\text{g}/\text{m}^3 \times 0.5 \times 22.5 \text{ hr}/24\text{hr}) \\
 &= 0.0031 + 0.023 \\
 &= 0.026
 \end{aligned}$$

4.3.4 Lead in Drinking Water

Drinking water can be a significant source of human exposure to lead, in that the entire population will have some level of exposure. Studies in the United States and Great Britain have demonstrated that drinking water is a significant contributor to total lead uptake, either directly or through the diet (USEPA, 1986; RSC, 1986; ATSDR, 1988).

4.3.4.1 Lead Concentrations in Drinking Water

Lead is present in drinking water primarily because of contamination from the distribution system. Plumbing in private residences contributes the major portion through leaching from lead pipe service connections or from lead-based solder used in copper plumbing. The leaching of lead from solder is more pronounced in newer homes but may decrease over time as the solder becomes coated with insoluble material (Levin, 1987).

A number of investigations have been undertaken in Canada and in Ontario to determine lead levels in drinking water (Appendix D Section D-2.2.2). Estimation of an average lead concentration in Ontario drinking water is difficult because of the different sources of drinking water and the variability in measured lead concentrations. The following observations are considered to be significant:

- National surveys of drinking water in 71 Canadian municipalities were conducted in 1976 and 1977. Median lead levels were found to be less than 1 µg/L. Average levels in tap water (3 to 5 minute flushed samples) were less than 1 µg/L, with a range of 1 to 65 µg/L (Meranger *et al.*, 1979, 1981).
- Using an on-site, integrated pump sampler in 18 Montreal homes, lead concentrations ranging from 0.25 to 276 µg/L were found, with a median of 0.65 µg/L. This method sampled a subset of the water each time the tap was turned on (Meranger *et al.*, 1984).
- The Distribution System Surveys were conducted in 76 Ontario municipal water systems from 1981 to 1987. The reported mean median concentrations for overnight standing, random standing and flushed samples were 30 µg/L (range 1-84 µg/L), 28.5 µg/L (range 1-423 µg/L) and 7.8 µg/L (range 1-22 µg/L), respectively. Residences with lead services had lead concentrations one order of magnitude higher than those that did not. However, elevated lead concentrations were not always associated with the presence of copper plumbing and lead/tin solder (Graham, personal communication, 1988).

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- The Duplicate Diet Study included drinking water samples collected in five Canadian cities. Concentrations ranged from 0.25 to 71.2 µg/L, averaging 8.8 µg/L with a median of 2 µg/L (Dabeka *et al.*, 1987).
 - In the Northern Ontario Blood Lead Study, flushed tap water samples demonstrated a mean median level of 3.3 µg/L, with a data range of 1 to 180 µg/L (Goss, Gilroy and Associates, 1989). Random and overnight standing samples showed slightly higher levels with mean median values of 7 µg/L (range 3-150 µg/L) and 6.6 µg/L (range 3-120 µg/L).
 - A 1988 survey of seven Ontario municipalities, employed composite samplers in 42 homes over a one week period. The average lead concentrations ranged from 1.1 to 30 µg/L, with a median of 4.8 µg/L (unpublished report).

4.3.4.2 Estimated Daily Intake of Lead in Drinking Water

Health and Welfare Canada has estimated that about 1 to 3% of daily lead intake, for both adults and children, comes directly from water. There is little difference between urban and rural residences. More recent analysis, however, suggests that this figure may be closer to 8% for adults. The Canadian Duplicate Diet Study found that water contributed 4.1% (mean value) or 1.4% (median value) of the total lead intake. For some individuals, who submitted water samples containing higher lead concentrations, drinking water contributed as much as 26% to total lead intake (Dabeka *et al.*, 1987).

In order to estimate the average daily consumption of lead in drinking water, a best approximate "average" lead concentration must be selected. A value of 5 µg/L was chosen, based on the 4.8 µg/L mean concentration found in the 1988 composite sampler survey. These data are considered to be most representative for the following reasons:

- The seven municipalities were selected to reflect major population areas and communities where representative lead levels had been found or were expected;
- The results from the composite samples were usually much more consistent than grab samples, especially where elevated levels were found; and
- The composite sample was linked to the water-use patterns of the residents as it samples a portion of the drinking water actually consumed by the household.

The 5 µg/L value falls between the mean median concentration of flushed (3-4 µg/L) and overnight (6.1 µg/L) samples found in the Drinking Water Surveillance Program, and between the flushed (3.3 µg/L) and overnight levels (6.6 µg/L) of the Northern Ontario

Blood Lead Study. The selected value also reflects the decline in lead concentration in flushed samples.

Based on the average daily consumption rates given in Table 4.6, the average daily intakes of lead from drinking water are calculated to range from 3.0 µg/day (0.23 µg/kg bw/d) for children to 7.5 µg/day (0.11 µg/kg bw/d) for adults. The figures correspond with those determined in a similar analysis by the Health and Welfare Canada Committee on Drinking Water (HWC, 1990b). Other workers have estimated a daily intake of lead from drinking water to range from 0.59 to 0.85 µg/kg/day (Meranger *et al.*, 1984).

TABLE 4.6 ESTIMATED LEAD INTAKES VIA DRINKING WATER

AGE GROUP	AVERAGE DAILY CONSUMPTION (Litres)	AVERAGE LEAD INTAKE (µg/day)	AVERAGE LEAD INTAKE (µg/kg/day)
Infant			
breast fed	0	0	0
not breast fed	0.2	1.0	0.14
1-4 yr	0.6	3.0	0.23
5-11 yr	1.0	5.0	0.19
12-19 yr	1.2	6.0	0.10
20+ yrs	1.5	7.5	0.11

4.3.5 Lead in Soils and Dusts

Soils and dusts can be significant sources of lead exposure for humans. However, determination of the relative contribution of these pathways to total individual lead intake is problematic. Gaps and deficiencies exist in the information available on the concentrations of lead in soil or dusts and on the quantities of dirt consumed. There is also considerable uncertainty in quantifying the dynamic relationship between lead levels in air, soil and dusts. Nevertheless, quantitative estimates have been made in this section, because ingestion of soils and dusts is believed to be a major contributor to lead intake in very young children, particularly for those living in the vicinity of industrial point sources.

4.3.5.1 Lead Concentrations in Ontario Soils

Soil lead levels ranging from 150 to 3000 µg/g have been reported for Canadian inner city sites (Nriagu, 1986). Surveys and sampling conducted by the MOEE have provided data on soil lead levels in Ontario. Data are available for both rural and urban localities, including areas impacted by lead smelter emissions. A summary of the data collected between 1972

and 1982 from areas removed from industrial point sources indicated mean soil lead values as follows: urban, 123 µg/g (range 5-845 µg/g); small town, 73 µg/g (range 2-133 µg/g); and rural, 35 µg/g (range <5-360 µg/g) (Rinne, 1989).

Information is available for specific urban sites (population greater than 10,000). Studies undertaken in the mid-1970's found a background lead concentration in Toronto garden soil of 99 µg/g (geometric mean) and average soil levels in a downtown area of 482 µg/g (range 10-1450 µg/g) (Roberts *et al.*, 1974). Soil sampling conducted in more recent epidemiological studies found mean soil levels of 54 µg/g in Toronto, 72 µg/g in Windsor (MOH, 1985), 31 µg/g in Thunder Bay and 13 µg/g in North Bay (Goos, Gilroy and Associates, 1989).

Specific urban residential mean concentrations, derived from pooled 1972 to 1985 MOEE data are as follows: 121 (±142) µg/g for Toronto; 119 (±95) µg/g for Windsor; and 150 (±93) µg/g for Guelph (Appendix D). These cities have the most extensive sampling databases. For the City of Toronto, the 233 samples ranged in value from 5 to 845 µg/g, with an estimated 2% of properties exceeding the 500 µg/g MOEE guideline. Analysis was done on composite samples (10 to 12 samples per residence) taken at a depth of zero to 5 cm.

4.3.5.2 Lead Concentrations in Dusts

Adults and children are exposed on a daily basis to dusts in homes, streets, playgrounds or through routine contact with other surfaces. The nature of human lead exposure through the soil/dusts pathway has been reviewed (MOEE, 1986; Duggan and Inskip, 1985; USEPA, 1986, 1989a). American epidemiological studies have demonstrated significant correlations between lead concentrations in soils and dusts and blood lead levels in children (Charney *et al.*, 1983; Bornschein *et al.*, 1986; Rabinowitz *et al.*, 1985). The most probable environmental exposure pathway for young children is illustrated below. Lead exposure through direct ingestion of soil will also occur in some children.

soil lead → house dust → hand dust → ingestion → blood lead

Analysis of lead exposure through soil and dusts requires knowledge of the lead concentration in these media and of its intermedial distribution in air, soil and dust. Many factors will influence this distribution, including: time, particle size, climatic conditions, and surface water solubility of the soil matrix (USEPA, 1989a).

The major contribution to lead in dust is from lead in soil. The upper layer of soil, sometimes referred to as "soil dust", is subject to re-entrainment by wind and human activity. Thus, although soil lead is relatively immobile, it may serve as a continuous source of lead in outdoor and indoor, or household, dusts (USEPA 1985, 1989a).

Other sources of lead in dusts are direct deposition from the atmosphere; motor vehicle and industrial emissions; and the weathering or renovation of lead-containing paint work. Few

data are available on the relative contribution of each source to the lead concentration in dust. With the phase-out of lead in gasoline, motor vehicle emissions are of less significance.

The relative contribution of exterior and household dusts to human lead exposure will be influenced by the time spent outdoors or indoors. Airborne lead particles associated with exterior dusts are highly mobile and subject to redistribution by wind currents. Given continuous input, the lead concentration in street dusts will drop following rainfall and street cleanings, lead being removed in road drainage water, and will rise between rainfalls (USEPA, 1989a). The concentrations of lead in playground, street and soil dusts are reported to increase with proximity to stationary lead sources and with higher concentrations of lead in air (Roels *et al.*, 1980; Brunekreef *et al.*, 1981). Little information is available on street dust lead concentrations in Ontario. A mean outdoor lead dust concentration of 1000 µg/g has been reported for urban Toronto (Roberts *et al.*, 1975) and a range of 250-2000 µg/g suggested for Canadian street dusts (Nriagu, 1986).

The concentration of lead in household dusts can be affected by a number of variables, including: house cleaning practices; seasonal conditions; particle size; air lead concentrations; the permeability of the home to outdoor air; and the amount of dust/soil carried into the home on clothing, shoes and pets. In Canada, typical house dust lead levels of 50 to 400 µg/g have been reported in suburban locations (Nriagu, 1986). In 1973, house dust concentrations in a Toronto urban control area were measured at a mean of 845 µg/g (range 351-2010 µg/g) (Roberts *et al.*, 1975). More recent data on lead concentrations in household dust in Ontario and Canada are not available. Information on house dust lead concentrations is notably sparse, particularly with regard to spatial and temporal variations (Duggan and Inskeip, 1985). This, coupled with a lack of standardized methodology for representative house dust sampling and analysis, makes interpretation of dust data highly uncertain.

The intermedial distribution of lead in soil, dust and air has been analyzed (USEPA, 1989a). Studies that include measurements of lead in air and soil/dust concentrations were used to predict the rate of change of lead concentrations in soil and indoor dust as a function of ambient air lead levels. It was assumed that changes in air lead would be followed by corresponding changes in soil and house dust lead concentrations. The following long-term equilibrium relationships were developed:

$$\text{Soil Lead} = 53.0 + 510.0 (\text{Ambient Air Lead})$$

$$\text{Dust Lead} = 60.0 + 844.0 (\text{Ambient Air Lead})$$

The equations predict the ratio of soil to dust lead concentrations to be roughly 1:1.6, at ambient air concentrations between 0.05 and 1.0 µg/m³.

In the short term, for periods of several months or less, changes in ambient air lead levels were predicted to result in direct changes to dust lead levels, but with little or no change to soil lead levels. The data on the time scales for the soil and dust lead changes were

inconclusive. Lead in an undisturbed soil matrix persists, but soil lead concentrations in disturbed, especially urban, environments reflect surface deposition after a period of years. Interior dust lead concentrations respond to air lead changes over a period of weeks or months, depending on interior-exterior access and interior recirculation or removal of dust (USEPA, 1989a).

No 1990's data were found on lead concentrations in street or house dust. Dust levels are generally expected to decline because of the recent large declines in ambient air lead levels in Ontario urban areas. If the current average value for air lead in urban areas ($0.05 \mu\text{g}/\text{m}^3$) is used in the above equations, a corresponding dust level of $102 \mu\text{g}/\text{g}$ is obtained. The equations, however, do not take into account any input from lead-based paint.

4.3.5.3 Estimated Daily Intake from Soil/Dust

Insufficient data prevent the quantitative separation of lead exposure through soil as opposed to dusts. The exposures must therefore be considered together. House dusts have been taken as the surrogate exposure vehicle for all soil and dusts, for the following reasons:

- evidence points to the importance of dusts as a critical path of exposure;
- children spend a larger proportion of time indoors; and
- house dust lead concentrations may exceed soil lead concentrations.

The estimated daily intakes for different age groups are presented in Table 4.7. Data on dust lead levels are lacking but may be derived by applying the soil lead/dust lead ratio of about 1:1.6 to soil lead concentrations representative of urban areas in Ontario ($150 \mu\text{g}/\text{g}$). The calculated value of $240 \mu\text{g}/\text{g}$ for urban house dust lies in the mid-range of values reported in Canadian suburban household dust (Nriagu, 1986). This may be an overestimate as this figure is twice as high as the value of $102 \mu\text{g}/\text{g}$ obtained using the equilibrium relationship for dust and air lead levels (Section 4.3.5.2). Therefore a reduced value of $200 \mu\text{g}/\text{g}$ is used to account for a possible reduction in the direct contribution of air lead to dusts. In point source locations, soil and dust levels will be significantly higher.

The daily amount of soil and dust a child ingests will depend on age, childhood habits and lifestyle. Earlier estimates of $100 \text{ mg}/\text{day}$ have appeared in several documents (Lepow *et al.*, 1974; Drill *et al.*, 1979; NAS, 1980; USEPA, 1986). More recent studies utilizing non-absorbed elements as tracers have estimated the amount at $65 \text{ mg}/\text{day}$ (Binder *et al.*, 1986) or 90 mg (Clausing *et al.*, 1987). Age-related changes in soil ingestion have been calculated, using age-related changes in blood lead concentrations and mouthing behaviour (Sedman, 1987). Considerable uncertainty is associated with such estimates. For the present exposure assessment a soil/dust ingestion figure of $80 \text{ mg}/\text{day}$ was assumed for children one to four years old.

No empirical evidence is available to provide soil ingestion rates for older children and adults. Exposure scenarios for adults assume that adults ingest less soil than children, because of differences in behaviour and personal hygiene. For the purposes of the exposure assessment, adults were therefore assumed to ingest 20 mg/day. For infants less than six months no soil/dust ingestion is assumed.

TABLE 4.7 ESTIMATED DAILY EXPOSURE OF LEAD FROM SOIL AND DUSTS BY URBAN CHILDREN AND ADULTS

	AGE	
	Children (1-4 yr)	Older Children & Adults
TYPICAL SOIL LEAD CONCENTRATION ($\mu\text{g/g}$)	150	150
HOUSE-DUST LEAD CONCENTRATION ($\mu\text{g/g}$)	200	200
SOIL/DUST INGESTED (mg/day)	80	20
DAILY INTAKE ($\mu\text{g/day}$)	16	4
($\mu\text{g/kg/day}$)	1.23	0.057 (adults)

4.3.6 Lead In Consumer Products

Exposure to lead may result from the use of certain consumer products, including house wares. Products with high levels of lead may present an acute health risk, as evidenced by reported cases of clinical lead poisoning. It is difficult, however, to make quantitative estimates of the contribution of such exposures to daily intakes of lead.

Renovation of old homes where lead-based paints are present may result in lead exposure due to ingestion or inhalation of paint chips, dust or fumes. This is a particular hazard for children and pregnant women. In the United States, cases of clinical lead poisonings have been reported in children who were at home at the time of paint removal or "deleading" procedures. Recently, cases of lead poisoning in children ingesting dusts from old weathering paint or renovations have been reported in Toronto and other areas of Canada. Levels of lead up to 500,000 $\mu\text{g/g}$ have been reported in the dust from paint removal (Inskip and Atterbury, 1983). This form of exposure is among the most hazardous and probably

represents the greatest source of exposure for incidental lead poisoning. Lead on building surfaces can also weather and contribute substantially to soil levels on residential properties.

Lead may leach from improperly glazed ceramics and pottery or from the lip and rim area of decorated glassware (Wilson and Card, 1986). Consumption of food or beverages from such containers produces the lead exposure. Lead from an "overglaze" ceramic or decorated drinking glass is believed to leach out immediately. Leaching increases if ceramic containers are used to store acidic foods, such as orange juice or tomato sauce, over long periods. Fatal cases of lead poisoning as a result of such exposures have been reported (Klein *et al.*, 1970). Imported ceramics, in particular, may contain high lead levels. Children also may be exposed to lead through ingestion of lead-containing paint and coatings from toys and other objects.

Lead may be present in tea and coffee made from water boiled in kettles with a lead-soldered interior (Ng and Martin, 1977). Lead leaches into water more readily at higher temperatures. In Canada, chemical analyses of water from such kettles have found lead levels of 750 µg/L. There have been recent cases of lead poisoning in the Toronto area, resulting from the use of imported decorative kettles and urns.

Certain hobbies may result in lead exposure through ingestion or inhalation. Such hobbies include: stained glass work, pottery glazing, lead sculpturing, wire soldering or use of some types of art supplies (Wallace and Cooper, 1986). Exposure to lead from ammunition is possible for gun enthusiasts using firing ranges or hunters packing shotgun shells at home. Imported cigarettes may contain lead from the use of lead arsenate pesticides on tobaccos.

Few data are available on lead exposure as a result of using certain consumer products. It is not possible to make scientifically defensible estimates of the daily intakes or relative contribution of these sources. However, because consumer goods can contribute to the total daily lead intake, they should be considered as an important component of the total exposure picture for individuals.

4.3.7 Integrated Exposure Estimates and Relative Contributions of Exposure Pathways - General Ontario Population

Integrated estimates of the total daily lead intake for various Ontario age groups may be derived from the summation of individual pathway point estimates. The results presented in Table 4.8 provide a quantification of the central tendency of exposure for these population groups and does not represent the complete range of exposures that may be encountered. Some of the factors which would increase exposure above these average estimates are assessed under special scenarios in the following section. Total multimedia intake of lead is estimated to range between 0.67 µg/kg/day and 2.87 µg/kg/day depending on age. In general, infants and young children will have higher intakes of lead than adult groups although the pathway contributions will differ. The typical young child has an estimated

total intake of 37.3 $\mu\text{g/day}$ or 2.87 $\mu\text{g/kg/day}$. This is roughly four times the adult dose on a per body weight basis, although the total intake in $\mu\text{g/day}$ is similar. Among infants, those that are breast fed have roughly 50% lower exposure than those fed formula or cow's milk. As discussed under dietary lead estimates, current dietary exposures may be reduced over those estimated on the basis of 1985 data. If adjusted on this basis the total integrated exposure for children (1-4 years) is estimated at 25.6 $\mu\text{g/day}$ (1.92 $\mu\text{g/kg/day}$) and for adults at 24.5 $\mu\text{g/day}$ (0.35 $\mu\text{g/kg/day}$) in 1993.

The relative contributions of food, drinking water, air and soil/dust exposures to total intake are shown in Table 4.9. For young children, food and soil/dust exposures account for the major portion of total intake at 47% and 45 % respectively. If as discussed under dietary exposures, the food intake values from 1985 data overestimate current exposure to some extent then soil/dust likely contribute a more significant proportion of exposure. Non-breast fed infant exposure is dominated by dietary exposure, whereas for the breast-fed infant, dirt/dust exposure may constitute the most significant exposure (if this occurs).

A different picture emerges for adults, with as much as 80% of exposure through diet and only 10% through dust intake. For all age groups, direct inhalation exposure is a minor pathway accounting for less than one-half of one percent of the young child total intake. The relative contribution of drinking water is approximately 8% in young children to 16% in adults under average conditions.

The dermal intake of lead is assumed to be trivial and is not estimated here because of the low dermal absorption of this metal.

The preceding information is integrated into an exposure "picture" for young children and adults (which include pregnant women and women of child-bearing age) by the multimedia exposure profile (MEP) illustrated in Figure 4.6 (based on actual 1985 foods data and on projections to 1993).

TABLE 4.8 INTEGRATED ESTIMATES OF LEAD INTAKE IN FOOD, WATER, AIR AND SOIL/DUSTS FOR VARIOUS AGE CLASSES OF AVERAGE URBAN ONTARIANS ($\mu\text{g/kg bw/day}$)

SUBSTRATE/MEDIA	INFANTS		CHILD (0.5-4 years)	CHILD (5-11 years)	TEENAGER (12-19 years)	ADULT (20-70 years)
	Breast Fed	Not Breast Fed				
Food	0.11	1.7	1.4	0.86	0.48	0.57
Drinking Water	0 ¹	0.14	0.23	0.19	0.10	0.11
Air	0.01	0.01	0.01	0.03	0.02	0.01
Soil/Dust	1.0	1.0	1.23	0.26	0.07	0.06
TOTAL DAILY INTAKE	1.12	2.85	2.87	1.34	0.67	0.75
Exposure Period (years)	0.5	0.5	3.5	6	7	50

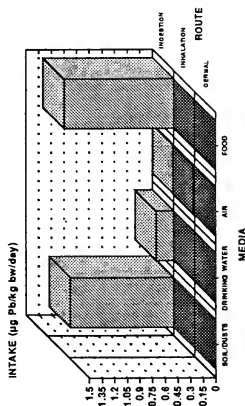
¹ exclusively breast fed, no drinking water supplementation

TABLE 4.9 RELATIVE CONTRIBUTIONS OF FOOD, WATER, AIR AND SOIL/DUSTS FOR VARIOUS AGE CLASSES OF AVERAGE URBAN ONTARIANS¹

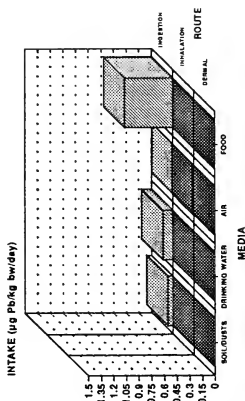
SUBSTRATE/ MEDIA	INFANTS		CHILD (0.5-4 years)	CHILD (5-11 years)	TEENAGER (12-19 years)	ADULT (20-70 years)
	Breast Fed	Not Breast Fed				
Food	9.8	59.6	48	64.2	71.7	76
Drinking Water	-	4.9	8	14.2	14.9	15
Air	0.9	0.35	<1	2.2	3	1
Soil/Dust	89.3	35.1	43	19.4	10.4	8
TOTAL	100	100	100	100	100	100

¹ Expressed as percentage (%)

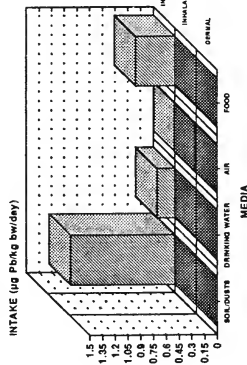
Child
Mid-late 1980's



Adult
Mid-late 1980's



Child
1992 (based on dietary projection)



Adult
1992 (based on dietary projection)

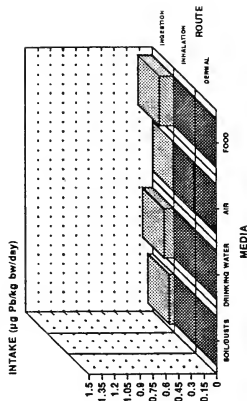


Figure 4.6 Multimedia Exposure Profile (MEP's) in Ontario Populations

In order to more fully understand the multimedia exposure assessment exercise, it is also important to examine the dynamic changes which have occurred in exposure during the last decade as a result of centralized regulatory mechanisms such as the phase-out of lead in gasoline and improvements in food canning processes in Canada. This is first reflected in declining blood lead levels in relatively few cross-sectional samples of children in the Toronto area between 1984 and 1990. To examine this further from the exposure modelling perspective, estimates for pre 1985, the mid-late 1980's and current estimates are calculated (Table 4.10 and 4.11). The total intake figures suggest that exposures have dropped by at least 50% in children and 25% in adults. This decline may have been greater if food levels have continued to decline similar to American levels. The differences in the estimates are due to changes in the general environmental levels of lead in these media during this period, particularly lead in foods and air. While overall intakes have declined, the relative contribution pattern has remained static for adults and changed somewhat for children. In the mid-1980's, each of lead in food and soil/dust were estimated to account for approximately 50% of total lead exposure in children. Soil/dusts are now probably the greatest exposure pathway given that the food values are likely to be overestimates.

TABLE 4.10 TREND ANALYSIS-INTEGRATED LEAD EXPOSURE ESTIMATES FOR URBAN ONTARIO POPULATIONS

MEDIUM	early 1980's		mid-late 1980's		1993 (based on dietary projection)	
	CHILD (1-4 YEARS) (µg/day)	ADULT (20+ YEARS) (µg/day)	CHILD (1-4 YEARS) (µg/day)	ADULT (20+ YEARS) (µg/day)	CHILD (1-4 YEARS) (µg/day)	ADULT (20+ YEARS) (µg/day)
Food	37.4	53.8	17.7	40	6	12
Drinking Water	3.0	7.5	3.0	7.7	3.0	7.5
Air	0.3	1.3	0.13	0.7	0.13	0.7
Soil/Dust	32.0	8.0	16	4.2	16	4.2
ESTIMATED TOTAL (µg/day)	72.7	70.6	36.8	52.6	25.1	24.4
(µg/kg/day)	5.6	1.0	2.8	0.75	1.9	0.35

TABLE 4.11 TREND ANALYSIS-RELATIVE CONTRIBUTION OF MEDIA TO TOTAL EXPOSURE (%)

MEDIUM	early 1980's		mid-late 1980's		1993 (based on dietary projection)	
	CHILD (1-4 YEARS)	ADULT (20+ YEARS)	CHILD (1-4 YEARS)	ADULT (20+ YEARS)	CHILD (1-4 YEARS)	ADULT (20+ YEARS)
Food	51	76	48	76	24	49.2
Drinking Water	4	11	8	15	11	30.7
Air	<1	2	<1	<1	<1	2.9
Soil/Dust	44	11	43	8	64	17.2
TOTAL	100	100	100	100	100	100

4.4 SPECIAL SUBPOPULATION ANALYSIS AND SCENARIOS

In addition to considering intakes for typical Ontario populations, exposure estimates were developed for other groups which may encounter greater exposure because of locale, behavioural characteristics and specific product use. These special scenarios include the following: residence near a point source of lead or in an area with high plumbosolvency; consumption of home grown vegetables from lead-contaminated soils; high incidental levels of soil/dust ingestion; infants fed formula from lead-soldered cans; consumption of acidic drinks from lead-glazed containers; and contact with lead-based paints. It is recognized that occupational exposure also presents a special exposure population, but this is not examined here. The estimated total daily intakes under such exposure scenarios are presented in Tables 4.12-4.14 and Figures 4.7-4.9. Assumptions and specific considerations for each analysis are outlined below. Estimates are presented for children and adults principally with inclusion of the dietary estimates based on 1985 total diet lead data. The impact on these figures of alternately assuming the projected 1993 dietary intakes (for the child and adult groups) on these estimates is discussed below.

Infant exposures are primarily influenced by diet, as suggested by the analysis for breast-feeding versus non-breast feeding. Consumption of canned milk stored in lead-soldered cans can greatly increase exposure to lead. Living in the vicinity of a point source of lead is likely to increase lead exposure incrementally: approximately 30% for a breast-fed infant. However, this is smaller for infants who do not live in the vicinity of a smelter and are fed cow's milk and other foods. Total exposure for infants (not breast-fed) is notably increased in areas of high plumbosolvency (3.56 µg/kg/day). The very high exposure calculated for drinking contaminated juices is an obvious hazard. Recently there have been reports of cases of

clinical lead poisoning in Toronto infants who were fed milk heated in imported Persian kettles containing lead solder.

For young children (0.5-4 years), living in the vicinity of a point source of lead is estimated to increase intake by roughly 25%. This agrees well with the differences in geometric mean blood lead levels of children living in the South Riverdale neighbourhood and control groups. Children in the smelter area exhibited slightly higher mean blood leads. Living in an area of high plumbosolvency is predicted to result in total daily exposures of 4.0 $\mu\text{g}/\text{kg}/\text{day}$, which is 40% greater than the average urban child and also greater than the intake predicted for living in the vicinity of a point source. Interestingly, if the model is reliable, consuming home-grown produce from typical urban soils also results in a total daily intake equivalent to either the point source or high plumbosolvent drinking water scenario. The significance of this pathway may have been previously underestimated, particularly for highly contaminated areas. High soil ingestion rates may result in exposures twice the typical daily intake. The paint ingestion and glazes scenarios are based upon the current federal regulatory limits. The actual levels in paints and glazes, particularly in imported materials, may be much higher. Even these conservative calculations indicate a high hazard associated with these exposures. The calculations suggest that such consumer products can result in exposures many times greater than all other exposures combined. This also reinforces the identification of historical lead-based paints on and in buildings as a potentially significant source of lead exposure for children.

Reduction of the background dietary exposure estimate based on the 1993 projected values would not effect the relative increases in intake ascribed to these situations. The net total exposure estimates would be reduced by roughly 0.8 $\mu\text{g}/\text{kg}/\text{day}$ to the following: average urban- 2.07 $\mu\text{g}/\text{kg}/\text{day}$; point source - 2.75 $\mu\text{g}/\text{kg}/\text{day}$; high plumbosolvency - 3.2 $\mu\text{g}/\text{kg}/\text{day}$; high soil ingestion 5.46 $\mu\text{g}/\text{kg}/\text{day}$; homegrown produce - 2.79 $\mu\text{g}/\text{kg}/\text{day}$. Because of the large predicted exposures associated with paint ingestion, the reduction in background dietary intakes is not considered a significant impact for this exposure.

Adult exposures under the various scenarios are generally much lower on a body weight basis than for children. This group also includes pregnant women and women of child-bearing age. Living near a point source or consuming home-grown vegetables results in a total daily intake only marginally greater than the average urban exposure. Living in a highly plumbosolvent area increases the predicted exposure to roughly 1.3 $\mu\text{g}/\text{kg}/\text{day}$ or 70% greater than average. High soil ingestion also increases exposures by a similar amount. Again, the hazard of drinking acidic juices from lead-glazed pottery is clear, particularly for pregnant women. Application of the projected 1993 dietary estimates to this data would reduce each total exposure by 0.4 $\mu\text{g}/\text{kg}/\text{day}$.

TABLE 4.12 INTEGRATED ESTIMATES OF LEAD INTAKE IN ONTARIO INFANTS SUBJECT TO VARIOUS EXPOSURE FACTORS ($\mu\text{g Pb/kg bw/day}$)

SUBSTRATE/ MEDIA	AVERAGE URBAN		FED CANNED MILK (LEAD SOLDER) ³	LIVING NEAR A POINT SOURCE ^{4,5}	LIVING IN AN AREA OF HIGH WATER PLUMBOSOLVENCY ^{2,6}	DRINKING JUICE FROM LEAD GLAZED POTTERY ⁷
	Breast Fed ¹	Not Breast Fed ²				
Food	0.11	1.7	5.3	0.11 ¹	1.7 ²	0.11
Drinking Water	0	0.14	0	0	0.85	0
Air	0.01	0.01	0.01	0.06	0.01	0.01
Soil/Dusts	1.0	1.0	1.0	1.48	1.0	1.0
Consumer Products	-	-	-	-	-	1) 10.00 2) 100.0
TOTAL DAILY INTAKE	1.12	2.85	6.31	1.65	3.56	1) 11.1 2) 101.1

¹ Assumes breast-fed only

² Fed cow's milk

³ Dabeka and Mackenzie, 1988

⁴ Based on long-term equilibrium of air/soil/dust relationship, mean monthly 1991 geometric mean level $0.28 \mu\text{m}^3$ lead predicted soil/dust = $566 \mu\text{g/g}$

⁵ Assume concentrations representative of combined soil and dust exposure.

⁶ Exposure calculated on flushed samples in MOEE composite survey upper end of range of values for average ($30 \mu\text{g/L}$) lead concentrations in the study was selected.

⁷ Assume 100 mL imbibed and concentrations of 1) 10% of current 7 mg/L limit; 2) 100% of current limit

Lead Exposure Scenarios for Infants (0 - 6 months old)

FIGURE 4.7

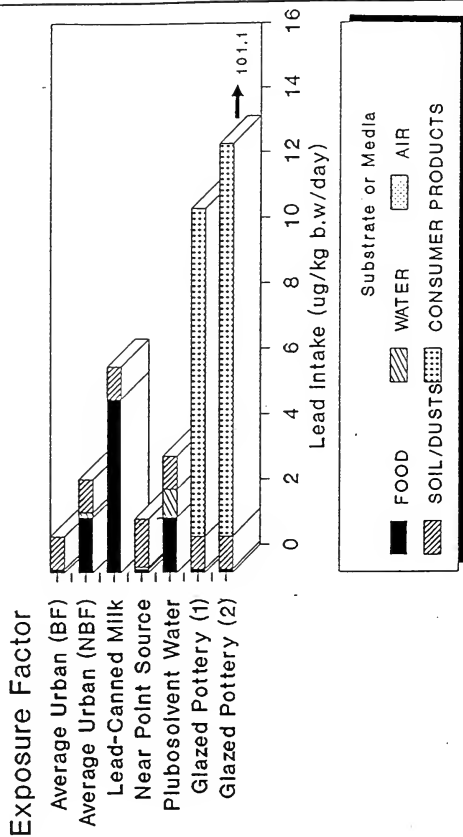


TABLE 4.13 INTEGRATED ESTIMATES OF LEAD INTAKE IN ONTARIO CHILDREN (0.5-4 years) SUBJECT TO VARIOUS EXPOSURE FACTORS ($\mu\text{g Pb/kg bw/day}$)

Substrate/ Media	Average Urban	Living Near a Point Source ¹	Living in an Area of High Plumbosolvency	High Soil Ingestion ²	Consuming Homegrown Produce ³	Paint Ingestion ⁴	Drinking Juice from Lead- glazed Pottery
Food	1.4	1.4	1.4	1.4	1.19 (other) 0.93 (homegrown)	1.4	1.4
Drinking Water	0.23	0.23	1.38	0.23	0.23	0.23	0.23
Air	0.01	0.12	0.01	0.01	0.01	0.01	0.01
Soil/Dusts	1.23	1.8	1.23	4.62	1.23	1.23	1.23
Consumer Products	-	-	-	-	-	1) 30.76 2) 3.07	1) 13.46 2) 134.6
Total Daily Intake	2.87	3.55	4.02	6.26	3.59	1) 33.63 2) 5.94	1) 16.33 2) 137.5

¹ Average point source value, mean of monthly geometric means air lead is $0.28 \mu\text{g Pb/m}^3$ (range 0.08-0.59). Predicted soil/dust level is $296 \mu\text{g/g}$.

² Assume $300 \text{ mg soil/dust ingested}$

³ Food concentration modelled from $150 \mu\text{g/g}$ soil concentration. Average contribution of vegetables (garden variety) to typical food exposures subtracted to avoid double counting.

⁴ Assume paint dust equivalency to house dust or $80 \text{ mg paint chips ingested}$ at concentrations of 1) 5000 and 2) $500 \mu\text{g/g}$.

FIGURE 4.8. Lead Exposure Scenarios for Children
(0.5-4 years old)

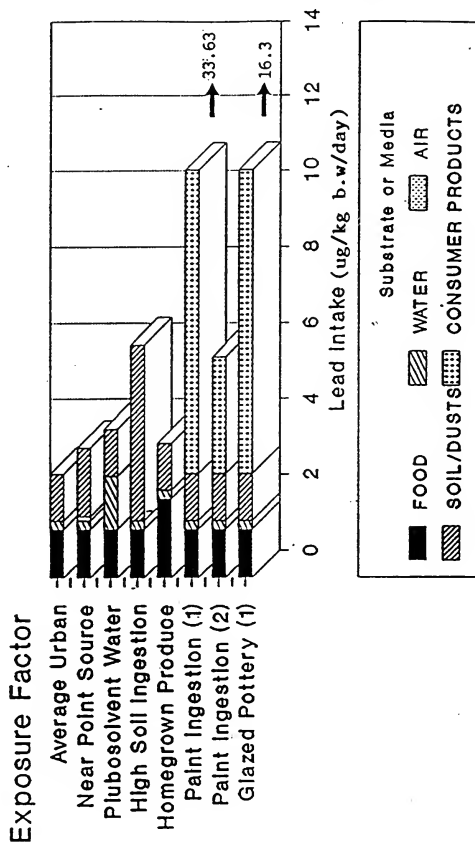


TABLE 4.14 INTEGRATED ESTIMATES OF LEAD INTAKE IN ONTARIO ADULTS SUBJECT TO VARIOUS EXPOSURE FACTORS ($\mu\text{g Pb/kg bw/day}$)

Substrate/Media	Average Urban	Living near a Point Source ¹	Living in an Area of High Plumbosolvency ²	High Soil Ingestion ³	Consuming Homegrown Vegetables ⁴	Drinking Juice from Lead Glazed Pottery ⁵
Food	0.57	0.57	0.57	0.57	0.50 (general) 0.23 (homegrown)	0.57
Drinking Water	0.11	0.11	0.64	0.11	0.11	0.11
Air	0.01	0.07	0.01	0.01	0.01	0.01
Soil/Dusts	0.06	0.09	0.06	0.6	0.06	0.06
Consumer Products	-	-	-	-	-	1) 3.0 2) 30
Total Daily Intake	0.75	0.84	1.28	1.3	0.9	1) 3.75 2) 30.75

¹ Assumes mean monthly geometric mean of $0.28 \mu\text{g}/\text{m}^3$ (range of means $0.08 - 0.59$) (MOEE, 1991)

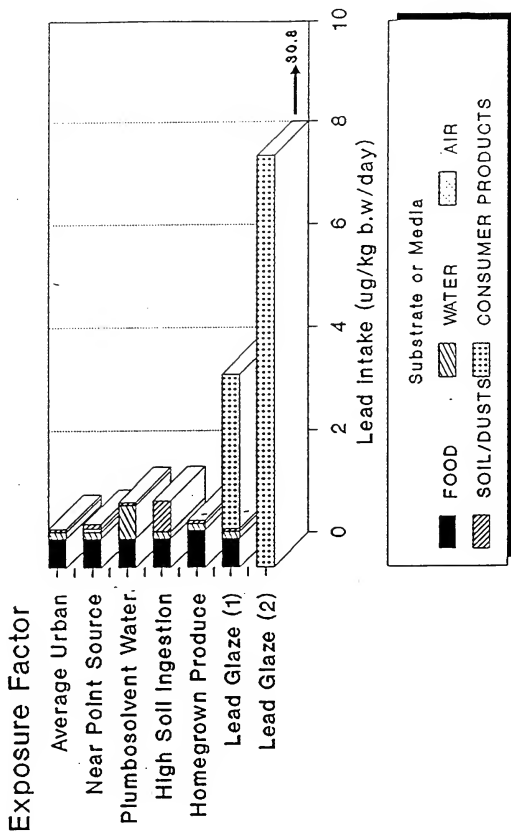
² Assumes drinking water concentration of $30 \mu\text{g}/\text{L}$

³ Assumes 200 mg/day soil/dust ingested through gardening or house cleaning activities

⁴ Assumes average urban soil concentrations ($150 \mu\text{g}/\text{g}$)

⁵ Assumes 300 mL imbibed at 1) 10% of $7 \text{ mg}/\text{L}$ federal limit 2) at 100% of $7 \text{ mg}/\text{L}$ federal limit

FIGURE 4.9 Lead Exposure Scenarios for Adults



4.4.1 Backyard Vegetable Consumption

In developing a soil guideline for lead, one must consider the exposure that might occur as a result of eating vegetables grown in lead-contaminated residential soils. Modelling this pathway requires the following information:

- the measured or predicted fresh weight concentration of the vegetables in question; and
- an estimate of the consumption rates for home grown produce.

The estimated human intake is given by the algorithm,

$$Intake = C_{food} \times IR$$

where C is the contaminant concentration in food (fresh weight, mg/kg) and IR is ingestion rate in kg/day.

The yields of various crops generally range from 0.3 to 2.6 kg/m². For a typical mixture of crops found in a backyard garden, a yield of 1.4 kg/m² is selected, although higher yields are possible with special techniques or extraordinary effort. The model assumes that the garden area is 30 m² with a yield of 1.4 kg/m². The total is 42 kg, which represents 13% of the vegetables and fruits that one adult and one child would consume in one year. For a family of four, approximately 7% of fruits and vegetables would be garden-grown, assuming that all the produce is consumed.

Nutrition Canada Survey data for Ontario indicate the average daily consumption of fruits, fruit products and vegetables is 372 g/day (26% of total diet) for young children (1-4 years) and 489 g/day (32% of total diet) for adult males; these values convert to 136 kg/year and 178.5 kg/year, respectively. In Ontario, the average leaf and root vegetable consumption (excluding potatoes) is 152 (144-180) grams/day for male adults; 83 (63-131) grams/day for female adults and 49 grams/day for children (1-4 years) (NCS, 1972). Average Ontario intakes for potatoes and other vegetables, excluding fruits, are approximately 98 grams/day for children (1-4 years) and 326 g/day for adult males (20-39 years). If 7% of the fruit and vegetables consumed are home grown, then the average consumption of home grown produce would be 26 g/day for children (1.8% of diet) and 34 g/day for adults (2.2% of diet) averaged over one year.

Predicted concentrations in foodstuffs at varying soil concentration are provided in Table 4.15 together with estimates of intakes for children and adults. Two models are presented: Model 1 based on "averaging" of the backyard crop exposure over a one-year period (i.e 26 g/day); and Model 2 based on all of the average daily "potatoes plus other vegetables" intake from the garden on a particular day. Fruits are not included. The second scenario is provided to account for those days, particularly during the growing season, when all of the daily intake of vegetables is from the garden. For example, on a particular day, a child may consumes a

meal consisting of roughly 100 grams of garden-grown potatoes and carrots. For an adult this may be 300 grams at one meal.

Predicted concentrations are derived using the uptake factors derived from recent studies on vegetables grown in lead-contaminated soil in the Toronto area (Bisessar and McIlveen, 1991). The concentration ratios or uptake factors (plant, dry weight/soil, dry weight) range from 2.5×10^{-3} in cabbage shoots to 7.6×10^{-2} in lettuce leaves. Edible portions of lettuce contain lower concentrations than root portions. Other values from the literature include 5.1×10^{-2} in blueberries growing on boreal soils (Sheppard and Evenden, 1990) and 7.4×10^{-3} in radish root (Sheppard and Sheppard, 1991). A mid-point value of the regression slopes (3.9×10^{-2}) from the Bisessar and McIlveen study was selected as representative of the uptake factor for backyard vegetables. This value agrees well with uptake factors for lead employed in other risk assessment exercises. To this value a dry weight to wet weight conversion factor of 0.085 was applied; this is the mid-range value of conversion factors across vegetable and fruit types (MOEE, 1990).

TABLE 4.15 PREDICTED DIETARY INTAKES OF LEAD VIA BACKYARD VEGETATION CONSUMPTION AT VARYING SOIL CONCENTRATIONS

SOIL LEAD CONCENTRATION ¹	UPTAKE FACTOR (PLANT/SOIL) ²	PREDICTED VEGETABLE/FRUIT CONCENTRATION ³	ESTIMATED DAILY INTAKE ⁴	
			Model 1	Model 2
50	3.9 X 10 ⁻²	0.16	0.32 (C) 0.08 (A)	1.23 (C) 0.68 (A)
100		0.33	0.66 (C) 0.16 (A)	2.56 (C) 1.36 (A)
150		0.49	0.98 (C) 0.24 (A)	3.69 (C) 2.04 (A)
200		0.66	1.32 (C) 0.32 (A)	5.12 (C) 2.72 (A)
300		0.99	1.98 (C) 0.48 (A)	7.68 (C) 4.08 (A)
400		1.32	2.64 (C) 0.64 (A)	10.2 (C) 6.80 (A)
500		1.65	3.30 (C) 0.80 (A)	12.8 (C) 8.80 (A)
750		2.47	4.94 (C) 1.20 (A)	19.2 (C) 10.2 (A)
1000		3.3	6.60 (C) 1.60 (A)	25.6 (C) 13.6 (A)

¹ µg/g ² dry weight ³ µg/g, FW ⁴ µg/kg/day; C=child, A=adult

4.4.2 Point Source Proximity

For this scenario, geometric mean soil and dust levels are derived from the long-term dynamic equilibrium relationships based upon the USEPA analysis of 40 community averages from different lead point sources, including 1980's data from two Toronto sites (USEPA, 1989a). The average relationships for the Toronto sites, provide coefficients somewhat greater than those of the aggregate equations. However, these coefficients were derived from data collected in the early 1970's when emission levels were substantially greater and prior to remedial measures. In the absence of more recent regressions, the aggregate relationships are utilized as air lead and surface soil concentrations from more recent measurements of other sites, which fall closer to the range of current air levels near these facilities. The aggregate has the advantage of representing a large collection of data in these relationships rather than relying on fragmented, site-specific information. As children in Ontario climates will spend significant portions of time indoors, the predicted dust levels are utilized as representative of total soil and dust ingestion, recognizing that house dust is partly derived from surface soil dusts.

Air lead concentrations for the scenario were chosen from the 1991 data of monitoring stations in the immediate vicinity of the Canada Metal facility (MOEE, unpublished data). The monthly geometric mean concentrations ranged from 0.08 to 0.58 $\mu\text{g}/\text{m}^3$. The mean of these, 0.29 $\mu\text{g}/\text{m}^3$, was selected as the typical exposure concentration for inhalation and for predicting soil and dust concentrations.

4.4.3 Areas of High Plumbosolvency

It is recognized that some areas of the province may experience somewhat higher concentrations of lead in drinking water. The mean median value of flushed samples from the 1981-1987 Ontario Distribution Survey of 76 municipalities was selected. This is considered to be a reasonable upper bound on concentration as the sites were representative of areas where higher lead levels would be expected.

4.4.4 Infant Food in Lead-soldered Tins

Storage of ready-to-use milk or formula in lead-soldered cans results in increased lead levels in these products. A 10-fold increase in lead intake was observed for infants, aged less than one month, who were fed such products compared to infants fed only cow or breast milk (Dabeka and McKenzie, 1988). Lead intakes of infants consuming cow milk, breast milk or glass-bottled infant formula were comparatively low, averaging 1.12, 1.11 and 1.26 $\mu\text{g}/\text{kg}/\text{day}$, respectively. However, infants fed canned ready-to-use formula had an estimated average intake of 5.3 $\mu\text{g}/\text{kg}/\text{day}$, with the lead from soldered cans accounting for approximately 77% of dietary lead. This is roughly three times higher than the IOC value suggested for lead. To the degree this type of product may have been phased-out of

Canadian markets since the time of the Dabeka and MacKenzie study, the likelihood of this exposure may have decreased.

4.4.5 Glazes - Food Storage

This scenario is presented using clinical literature reports on this type of lead-poisoning. The concentrations selected are based upon the current federal regularity leaching limit (under review) and the possible exposure allowed by 10% of this limit.

This scenario, although not of high incidence, is considered plausible given the use of imported vessels as well as the large number of hobbyists in Ontario who produce and sell such items, many of whom mix their own glazes.

4.4.6 Paint Ingestion

Lead-poisoning cases in Ontario children have been reported in association with both renovation of old homes as well as external weathering of lead-based paints. The current scenario is provided to assess the degree of exposure to dusts or chips of paint using the currently allowable lead limit in paints, as well as 10% of that limit. If one considers the erosion of paints to contaminate soil dusts or house dusts directly, the potential exposure pathway does not differ from direct soil/dust ingestion. Direct ingestion of chips of paint, as in a pica child, would contribute to even higher exposures. It should be pointed out that historically, lead paints have contained very much higher levels of lead so that the predicted exposure through release of such paint to soils or indoor environments through renovation, may lead to very significant exposures. This is further supported by some of the very high levels of lead in paints found in soils close to exterior walls of some homes in Toronto. The allowable level of lead in new paints was revised downwards to 6000 ppm in 1970 under the federal Hazardous Products Act and is currently under revision to 600 ppm. There is very little lead found in new paints and coatings with the exception of road and safety paints which may contain lead chromate for bright yellow markings.

4.4.7 High Soil Ingestion

Some young children, depending on hygiene, play habits and inclination may ingest greater than average amounts of soil. For instance, vigorous play in a sandbox area may lead to much higher soil ingestion for a particular child. A value of 300 mg/day (roughly four times the average) was selected as a plausible high soil ingestion rate for a normal child. It falls well within the range of values suggested by individual tracer element studies (Calabrese *et al.*, 1991). This is not meant to represent pica or geophagic behaviour which are special behavioural conditions in children requiring specific medical attention. Even these figures

may underestimate exposure for children who play very vigorously in soil, for instance, in a public playground.

SUMMARY

- The multimedia approach which considers total exposures from all environmental media was used to evaluate human exposure to lead. The estimates arrived at appear roughly congruent with the results of epidemiological surveys of blood lead levels in Ontario child populations.
- Based on epidemiological surveys and an analysis of integrated pathway exposures, a considerable reduction in general lead exposure has taken place in Ontario since the mid-1980's. In 1990, blood lead levels in Toronto children, removed from an industrial point source, averaged approximately 4 $\mu\text{g}/\text{dL}$. A significantly higher mean of 12.0 $\mu\text{g}/\text{dL}$ was found for urban children in the 1984 Ontario Blood Lead Study. Estimated intakes of lead for young children, based on a multimedia model, have declined by at least 50% over the same period. The declines may be attributed to the phasing-out of lead in gasoline and voluntary elimination of lead solder in food canning processes.
- Daily lead intakes from all major pathways for urban children are estimated to be 37 $\mu\text{g}/\text{day}$ or 2.87 $\mu\text{g}/\text{kg}/\text{day}$. The corresponding adult intake (which includes pregnant women) is estimated at 53 $\mu\text{g}/\text{day}$ or 0.75 $\mu\text{g}/\text{kg}/\text{day}$. The exposure profile for children indicates that intakes from food represent about 48% of exposure (17.7 $\mu\text{g}/\text{day}$ or 1.4 $\mu\text{g}/\text{kg}/\text{day}$). Based on trend analysis, however, this may be an overestimate. Exposure from soil and dust was quantitatively assessed using observed and projected trends in soil and dust levels based on recent reductions in ambient air lead levels. Although use of leaded gasoline has declined markedly, previous use has resulted in widespread contamination of soil and dust. An average daily intake of 16 $\mu\text{g}/\text{day}$ or 1.23 $\mu\text{g}/\text{kg}/\text{day}$ is estimated for children or about 43% of total intake. Drinking water represents about an average of 8% of total intake (3 $\mu\text{g}/\text{day}$ or 0.23 $\mu\text{g}/\text{kg}/\text{day}$). Finally, direct inhalation of lead, is a relatively minor direct exposure route estimated at 0.13 $\mu\text{g}/\text{day}$ or 0.01 $\mu\text{g}/\text{kg}/\text{day}$. Dermal absorption of lead is negligible. This information is utilized to develop a Multimedia Exposure Profile (MEP) for young children and adults.
- Estimated daily intakes of lead from food, based upon the summation of 112 food grouping intakes were developed. Highest total intake increases with age from 17.7 $\mu\text{g}/\text{day}$ in young children to approximately 40 $\mu\text{g}/\text{day}$ in adults. However, infants and children are expected to have the highest exposure on a body weight basis at 1.6 and 1.4 $\mu\text{g}/\text{kg}/\text{day}$, respectively, compared to adult values of 0.6 $\mu\text{g}/\text{kg}/\text{day}$. These dietary lead intakes are based upon dietary data collected in Canada in 1985. Based on the observed significant declines in blood lead which have been observed, the post 1990 phaseout of lead in gasoline, voluntary changes in the food processing industry and observed trends in the U.S. it is projected that dietary intakes may have declined from roughly 18 $\mu\text{g}/\text{day}$ in the mid 1980's to 6 $\mu\text{g}/\text{day}$ in 1993. This estimate is limited as it is not based on actual analysis of current food baskets. Current total

daily intake estimates for 1993 children may be 1.92 µg/kg/day and 0.35 µg/kg/day for adults.

- Some earlier estimates indicate that women of child bearing age (20-39) years are likely to have significantly lower intakes than men. Therefore the average adult value of 0.6 µg/kg/day is probably an overestimate for this group. In the last Nutrition Canada Survey, pregnant women had a pattern of food consumption similar to that of 20 to 39 year-old females, except for modest increases in milk and fruit consumption. Estimated lead intakes would not differ significantly and would be unlikely to exceed 0.6 µg/kg/day on average.
- The analysis indicates that exposure from breast milk is small and likely less than that associated with formula or mixed diets. Infants fed formula from lead-soldered cans may encounter much higher exposure.
- There are differing scenarios which may result in higher exposures for certain subpopulations. These may be either chronic or intermittent acute exposures which occur against the background of typical exposure. For example, urban children living in the vicinity of industrial point sources of lead emissions or children living in areas of higher plumbosolvency are expected to be more highly exposed. Estimates of total daily exposure suggest that children have potential exposures roughly 25% greater than typical urban children under the point source scenario and roughly 40% higher in areas of highly plumbosolvent drinking water.
- Exposure from consumption of home grown vegetables is assessed at various soil levels and it is concluded that this indirect route can contribute lead intakes as large as direct ingestion of soil. Therefore consideration of this exposure in developing soil-related guidelines would appear essential.
- For any child, exposure to lead through certain consumer products such as lead-based paints, ceramic glazes, or hobbyist materials can be potentially hazardous with a risk of clinical poisoning. These acute exposures occur against the "background" of chronic exposure described above. Modelled exposure scenarios indicate that such intermittent exposures may produce exposures much greater than all other environmental exposures combined. Renovation of homes containing lead-based paints can create conditions of high exposure for children and women of child bearing age in Ontario. Weathering or flaking of exterior lead-based paints may also lead to unsafe levels of exposure. The quantitative estimates provided here, together with anecdotal information and U.S. experience indicate that these intermittent exposures are therefore of particular concern for Ontario children, whether exposure is to the fetus or to toddlers.
- New sources of lead exposure are emerging. There have been recent cases of lead poisoning in Ontario resulting from the use of imported decorative kettles and urns.

5.0 ALTERNATIVE EXPOSURE MODELS

The following section briefly describes significant and emerging models of lead exposure in children, which are currently in development and use in some situations, particularly as related to lead contamination of soils. These approaches are reviewed as to their utility as an aid in standards development.

5.1 Integrated Uptake or Biokinetic Model of Lead Exposure

5.1.1 Description

The biokinetic model employs a somewhat more complex methodology than the deterministic approach. Such models incorporate considerations of absorption or uptake dynamics as well as the distribution kinetics of lead in various tissue compartments. The Integrated Uptake/Biokinetic Model (IU/BK), developed by Harley and Kneip (1985), has been used by USEPA in developing lead standards and guidelines (USEPA, 1990). It is being used by the USEPA Office of Air Quality Planning and Standards (OAQPS) in developing a revised National Ambient Air Quality Standard (NAAQS) for lead. It also has been adapted by the Environmental Criteria and Assessment Office (ECAO) as a potential method for determining clean-up levels for lead-contaminated soils.

It is important to note that there are various versions of the IU/BK model, the earliest (eg. version 2.0) of which was developed specifically for application to point source emitter situations as related to the question of a revised NAAQS's. More recently the model has found use in discussions regarding drinking water standards. Also a substantive revision and refinement of the model (version 4.0 and 5.0) has been undertaken by the Office of Solid Waste and Emergency Response (OSWER) to apply this model for developing soil lead clean-up levels at residential CERCLA/RCRA sites.

For the purposes of Ontario guideline development, this methodology is viewed as a useful adjunct to the multimedia model estimates. It may be used in examining scenarios for sets of general guidelines derived from the multimedia model in addition to site-specific situations, for which it was originally developed.

The model predicts total lead uptake for children aged up to seven years by using the lead concentrations in media associated with various exposure pathways (soil, dust, diet, water, air). The concentrations may be either monitoring data or levels obtained through modelling programs. These are then combined with age-specific intake (inhalation and ingestion) and uptake (absorption) parameters to predict total lead uptakes. The latter are then transformed to mean blood lead levels, which together with a geometric standard deviation, can be used to estimate the frequency distribution of blood lead levels in children (USEPA, 1989a). Blood lead is a valuable parameter for the purposes of lead risk characterization. Site-specific

source contributions to blood lead levels as well as contributions from all media may also be examined.

Although originally designed for site-specific situations, such as locations in the vicinity of a lead processing plant, broader applications may be feasible. The model is flexible in that it allows for site-specific input data or non-site-specific default values. As new studies are conducted, the default values may be updated to reflect improved understanding of a given exposure factor. The default values used in the most recent version of the model for children aged two to three years are given in Table 5.1.

In this way, modelled impacts on predicted blood leads may be examined under a variety of scenarios. As a simple example, allow all exposure parameters except for soil/dust to be set at zero; in other words, there is no lead intake from air, water, or diet. The model predicts that soil clean-up lead levels would have to be set at 600 ppm in order to have 95% of the children with blood lead levels below 10 $\mu\text{g}/\text{dL}$.

This exercise would therefore roughly define the upper limit for soil clean-up levels based on current assumptions about soil/dust intake and uptake (Table 5.2). In addition, the required soil clean-up levels are given should the blood lead cut-off value be alternately set at 15 $\mu\text{g}/\text{dL}$. For the conditions modeled in Table 5.2, a soil/dust level which places 95% of children below 10 $\mu\text{g}/\text{dL}$, also has 99.9% below 15 $\mu\text{g}/\text{dL}$ level.

The model is most sensitive to the following factors:

- changes in the input soil/dust levels themselves;
- changes in the soil/dust ingestion rate;
- the bioavailability or soil/dust absorption value (or the default dust value if only soil levels are measured at a site); and
- changes in the diet values.

Changes in water or air values have limited effects on soil/dust clean-up levels due to their minor contribution to the total lead uptake. Similarly scenarios for deriving allowable levels in other pathways, such as drinking water, can be examined.

The model contains a large number of assumptions and may therefore be affected by individual parameters, such as absorption coefficients. For example, Table 5.3 shows how variations in the absorption value would affect desired soil levels. A similar analysis could be applied to soil ingestion rates. The model therefore may be used to test such assumptions through use of specific site data, like the South Riverdale clean up project.

Changing the geometric standard deviation (GSD) can also have major effects on the soil/dust clean-up level. Such changes do not alter the mean intake/uptake, as was accomplished by the changes described above, but alter the shape of the distribution curve. A low GSD signifies a high and narrow curve. A high GSD, by contrast, signifies lower

confidence in the geometric mean and the distribution curve is shorter and broader with a long tail in the high blood lead regions. GSD values typically range from 1.30 to 1.50, with an average of 1.42 for the studies listed. The default value of 1.42 matches the GSD found in the NHANES II study, and thus was the value chosen by USEPA as default for the model. Modelling of Ontario scenarios would require determination of GSD values from available and ongoing blood lead studies.

TABLE 5.1 INTEGRATED INTAKE/BIOKINETIC MODEL FOR LEAD

PARAMETER No.	DESCRIPTION	DEFAULT VALUE
AIR		
1	Outdoor air lead ($\mu\text{g}/\text{m}^3$)	0.2
2	Indoor air lead ($\mu\text{g}/\text{m}^3$)	0.06
3	Time spent outdoors (hour/day)	3
4	Time weighted average for air ($\mu\text{g}/\text{m}^3$)	0.10
5	Breathing volume (m^3/day)	5
6	Lead intake from breathing ($\mu\text{g}/\text{day}$)	0.5
7	% Respiratory deposition/absorption	32
8	Lead uptake from air ($\mu\text{g}/\text{day}$)	0.2
DIET		
9	Lead intake from diet ($\mu\text{g}/\text{day}$)	29
10	% Gastrointestinal absorption	50
11	Lead uptake from diet ($\mu\text{g}/\text{day}$)	14.5
SOIL		
12	Outdoor soil lead ($\mu\text{g}/\text{g}$ or ppm)	200
13	Indoor dust lead ($\mu\text{g}/\text{g}$ or ppm)	200
14	Daily soil-dust ingestion (g/day)	0.1
15	Weighting factors (soil/dust)	45/55
16	Lead intake from dust and soil ($\mu\text{g}/\text{day}$)	20
17	% Gastrointestinal absorption	30
18	Lead uptake from dust and soil ($\mu\text{g}/\text{day}$)	6.0
WATER		
19	Drinking water lead ($\mu\text{g}/\text{L}$)	9
20	Drinking water intake (L/day)	0.5
21	Lead intake from drinking water ($\mu\text{g}/\text{day}$)	4.5
22	% Gastrointestinal absorbance	50
23	Lead uptake from drinking water ($\mu\text{g}/\text{day}$)	2.3
PAINT		
24	Lead intake from leaded paint	0
TOTAL		
25	Total lead uptake ($\mu\text{g}/\text{day}$)	23

Default values: For two to three year old children exposed to lead in air, diet, dust, soil, and drinking water.
 Percentage contribution: 63% diet, 26% soil, 10% water, 1% air.

Several validation exercises have been completed on the original biokinetic model developed by OAQPS. These have involved comparison of predicted and observed blood lead levels of children living near point sources of lead. The most extensive study used 1983 data for 400 children, aged one to five years, living near a lead smelter in East Helena, Montana. Using site-specific data as input into the model, the actual and predicted mean blood lead values were nearly identical for children living within 2.25 miles of the smelter (USEPA, 1989a). Predicted soil and dust levels were also used to test the model when site-specific data are missing. The model predicted a blood lead value of 9.5 µg/dL whereas the observed mean value was 9.3 µg/dL. Other validations in Omaha, Nebraska, and Kellogg, Idaho, were less extensive, but support the East Helena results: the model performs well in predicting mean blood lead levels in children living near point sources of lead (USEPA, 1989a).

TABLE 5.2 REQUIRED SOIL/DUST CLEAN-UP LEVELS (PPM) TO KEEP 95% OF CHILDREN¹ BELOW GIVEN BLOOD LEAD LEVEL

MODELLING PARAMETERS	BLOOD LEAD LEVEL	
	10 µg/dL	15 µg/dL
Current model, all default	600	350
Default, diet value at 10 µg/day; all other parameters set to zero	350	640

¹Aged 2-3 years

TABLE 5.3 REQUIRED SOIL/DUST LEVELS (PPM) GENERATED USING DIFFERENT SOIL/DUST ABSORPTION VALUES¹

ABSORPTION VALUE	REQUIRED SOIL/DUST LEVEL	
	CUTOFF 10 µg/dL	CUTOFF 15 µg/dL
50	200	385
40	250	480
30	350	640
26	400	740
21	500	920
17	600	1130
15	700	1280
5	2000	3850

¹ 1990 diet values, all else default; 95% of children have PbB levels below cut-off values.

The current ECAO version of the biokinetic model is essentially the same as the original OAQPS model, but several of the parameters have been changed to more conservative values. The current model is being used at the large Bunker Hill Superfund Site in Idaho to determine appropriate clean-up levels for soil lead. Validations of the model were made for the years 1983 and 1989. Site-specific data were used, including soil, dust, diet, air and water lead concentrations as well as site-specific soil ingestion/absorption values and GSD values. This validation exercise found that the model over-predicted blood lead levels using the model default values for soil ingestion and absorption. Hence, site-specific dose coefficients (the product of the absorption value and ingestion rate) were run rather than default values.

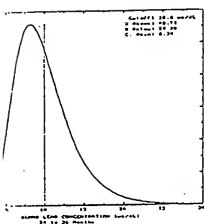
The environmental levels and media-specific consumption parameters of the deterministic multimedia model were used to generate predicted blood lead distributions in children through the IU/BK model. Default biokinetic slope factors and transfer coefficients were utilized. Calculated blood lead and media uptake values were generated (Table 5.4) (Figure 5.1). At a blood lead cut-off value of 10 $\mu\text{g}/\text{dL}$, the model suggests a dynamically changing exposure picture: large percentages were above this cut-off in the early 1980's, but relatively small, perhaps less than 1%, in the 1990's. This, of course, does not account for incidental exposures where individual blood leads may be notably elevated; for example, to lead-based paint or areas near point sources. The changes are driven by estimated reductions in dietary and airborne lead concentrations.

It is instructional to compare the predictions of the model with the epidemiological data on blood lead levels in Ontario children. The model predicts a geometric mean blood lead of 8.7 $\mu\text{g}/\text{dL}$ as opposed to the level of 12.02 $\mu\text{g}/\text{dL}$, observed in urban children in the 1984 Ontario Blood Lead Study (Duncan *et al.*, 1985). Better agreement is seen with later 1980's data, where the IU/BK model estimates approximately 6 $\mu\text{g}/\text{dL}$, as opposed to a mean of 7.87 $\mu\text{g}/\text{dL}$ observed in urban Northern Ontario children in 1987 (Goss, Gilroy, 1988). It compares well to the OBLS control blood lead for 1990 of 3.9 $\mu\text{g}/\text{dL}$.

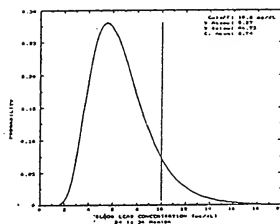
The differences may be due to the following:

- underestimation of exposures related to diet or soil dusts.
- underestimation of absorption of ingested inorganic lead, or
- inability to account for incidental exposures related to lead-based paints or other common product sources.

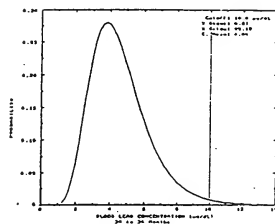
The degree to which this model may be applied to broader populations, as opposed to site-specific questions, requires further analysis and consideration.



early-mid 1980's



Late 1980's



1997

Figure 5.1 Predicted Blood Lead Distributions in Children

5.1.2 Evaluation

The IU/BK model represents a distinct attempt to develop a conceptually sound approach to human exposure assessment for lead. It involves a consideration of a variety of pathway exposures as well as an effort to transform predicted intake values to estimates of resultant blood lead distributions in sensitive populations.

While conceptually sound, the model contains a large number of variable term coefficients and is very dependant on the use of default specifications that would be used when suitable site-specific data were not available (e.g dietary lead input). The reliability of the results provided is therefore subject to question. Situations for which the use of default values is not appropriate and where sampling or measurement should occur are not sufficiently defined

Our experience is that this model is particularly sensitive to the choice of geometric standard deviation (GSD) of blood lead used. To what degree the default GSD of 1.4 is applicable to Ontario situations is uncertain and no guidance is provided as to the limitations on the size of population that can be considered. Certainly the user will in general not be sufficiently informed regarding community blood lead data or such data may not exist to select an appropriate GSD. In a simplistic comparative analysis, the model significantly underestimated mean blood lead level when compared to measure Ontario urban population means. The model may not be able to adequately predict variation in exposure across a general population. This suggests that considerable caution should be exercised in application of the model in the absence of alternate analyses.

Applicability of the model to the larger population beyond site-specific application is not delimited although the model was originally fashioned for site-specific analysis. This implies that the model may be more generally applicable to larger population groups as opposed to geographically limited groups. This type of application would require further validation in that broader context.

TABLE 5.4 IU/BK MODEL: CALCULATED BLOOD LEAD AND RELATIVE MEDIA UPTAKES FOR CHILDREN (NON-POINT SOURCE)¹

TIME PERIOD	AGE	G.M. BLOOD LEVEL ²	DIET UPTAKE ³	WATER UPTAKE ³	SOIL/DUST UPTAKE ³	AIR ³	TOTAL ³
Early-mid 1980's	1-2	8.1	18.7	1.4	9.1	0.16	29.4
	2-3	8.4	18.7	1.4	9.1	0.27	29.5
	3-4	8.5	18.7	1.4	9.1	0.28	29.5
Late 1980's	1-2	6.3	9.1	1.4	9.1	0.05	19.6
	2-3	5.7	9.1	1.4	9.1	0.09	19.7
	3-4	5.7	9.1	1.4	9.1	0.09	19.7
1992	1-2	4.8	5.0	1.4	6.9	0.03	13.4
	2-3	4.6	5.0	1.4	6.9	0.05	13.4
	3-4	3.9	5.0	1.4	6.9	0.05	13.5

¹ Linear absorption methodologies utilised ² µg/dL ³ µg/day

5.2 Society for Environmental Geochemistry and Health Model (SEGH) for Lead in Soil

5.2.1 Description

The SEGH model was developed because of the regulatory confusion caused by the lack of a U.S. standard for lead in soil. A "phased action plan" for lead in soil was developed utilizing a matrix approach (Wixson, 1989). This was based on the presumption that a single number for all situations was unrealistic for various reasons, related mostly to land use. A soil-lead matrix standards was developed as a relationship or formula to allow for a variety of environmental situations (land use, exposed populations) and regulatory criteria (blood lead).

Unlike the biokinetic model which models blood lead, the SEGH model requires site-specific information regarding community blood lead concentrations and their distribution in children. In the SEGH model, blood lead concentrations is equated to a baseline or background level plus an increment due to soil and dust. The baseline is suggested to account for all other exposures. The slope of the blood-lead/soil lead relationship can vary depending on situation and can be modified as more site-specific data become available.

The suggested blood-lead/soil lead relationship is given by the following equation:

$$S = \frac{\left[\frac{T - B}{G^n} \right] \times 1000}{\delta}$$

where S = geometric mean soil lead concentration, ppm

T = target Blood Lead Level, µg/dL

G = Geometric Standard Deviation of Blood Lead Distribution

n = Number of corresponding standards deviations

B = Background or baseline Pb-blood

δ = slope of the blood lead/soil-lead relationship, µg Pb/dL per 1000 ppm (µg/g)

Through selection of a target blood lead and the percentage of the population to be protected, the level of lead in soil can be calculated. As an example, what would be the derived guideline where 99% of children were protected to a target blood lead concentration of 10 µg/dL in a population with a background blood lead of 4 µg/dL and a GSD of 1.5 ? From above, assuming δ=2 :

$$S = \frac{\frac{10 - 4}{1.5^3} \times 1,000}{2} = 520 \text{ ppm}$$

The variation in the suggested soil lead guideline will be considerable depending on the δ value selected as this is the largest area of uncertainty. Published values range two orders of magnitude, with a 'typical' range of 2-5. Examples of the variation in resultant guideline levels upon variation of factors is presented in Tables 5.5 and 5.6.

TABLE 5.5 DERIVED SEGH MODEL SOIL LEAD LEVELS DETERMINED BY THE TARGET BLOOD LEAD AND PERCENT OF POPULATION TO BE PROTECTED

Target Blood Lead Level ($\mu\text{g}/\text{dL}$)	Soil Standard (ppm) at % Population less than Target Blood Lead			
	50%	95%	99%	99.9%
10	3000	880	300	-
15	5500	2300	1400	700
20	8800	3750	2600	1600
25	10,000	5200	3700	2500

(adapted from Wixson, 1989)

TABLE 5.6 EFFECT OF VARIATION OF SOIL LEAD/BLOOD LEAD FACTOR AND TARGET BLOOD LEAD ON DERIVED SOIL STANDARDS FROM SEGH MODEL

Target Blood Lead Level ($\mu\text{g}/\text{dL}$)	Soil Standard (ppm) at various δ values ($\mu\text{g}/\text{dL}$ per mg/kg soil)			
	1	2	3	8
10	600	300	150	75
15	2900	1400	700	350
20	5200	2600	1300	650
25	7500	3700	1850	925

(adapted from Wixson and Davies, 1991)

5.2.2 Evaluation

The SEGH model overcomes one disadvantage of the IU/BK model in that it relies on empirical derivation and therefore the numerous assumptions regarding other exposures, soil ingestion, bioavailability, etc are not needed. Unlike the IU/BK model, this model has a requirement for information regarding site-specific community blood lead levels and distributions in children. This would likely limit this model to application to situations where there are inadequate numbers of children in an area from which to gather such statistics. This also may preclude its direct application to modelling various remediation scenarios or using predicted future site data. The results are also greatly effected by the selection of soil lead/blood lead slope factors. Similar to the overall model presented in this document, an advantage of the SEGH approach is that there is considerably less reliance on assumptions regarding lead bioavailability, biokinetic slopes and other factors. This model may be a useful adjunct to decision-making in site-specific situations where a more robust data is available. Validation exercises of this model would be valuable.

SUMMARY

- The USEPA biokinetic model and the SEGH lead in soil model are reviewed here with the aim of evaluating their possible use in assessing total lead exposure for children and as a potential aid to the Ministry in the guideline development process. Both models appear to have merits particularly as related to the issue of site-specific soil guidelines. There are some notable limitations and both models would benefit from a concurrent validation, particularly at an Ontario site. Both are viewed as useful complements to the deterministic modelling presented in this document. Use of these models as a comparison for derived health based-criteria (all media for the IU/BK; soil only for the SEGH model) is suggested.
- The SEGH model requires significant amounts of site-specific empirical data (blood leads and soil/blood lead relationships); the IU/BK model can be run on a variety of default values in the absence of specific data.
- The IU/BK and SEGH models yield discrepant results under conditions of typical assumptions (data not shown), with the SEGH models suggesting significantly lower soil guidelines for a given scenario.
- Based on the relative merits of these evolving models, it is recommended that they be utilized as an aid in the quantitative evaluation of standards. The IU/BK model should at this point be limited to a relative check of the alternative sets of standards proposed as opposed to quantitative derivation of guideline values based on the caveats described above. Both the IU/BK model and the SEGH model may be useful to site specific assessment of lead in soil, particularly if applied together.

6.0 RISK CHARACTERIZATION

The critical effects of lead are identified as potential neurobehavioural and developmental deficits associated with low level exposure. It appears that the developmental effects associated with prenatal exposure may be greater than those associated with postnatal exposure, but this remains to be assessed through later follow-up in the ongoing prospective studies. Characterization of risk therefore is examined as the likelihood of neurobehavioural deficits in various populations.

Although the effects of low-level exposure may not seem severe in the individual child, on a population basis they are nonetheless considered important. Because the health significance of these effects is discussed in terms of population effects rather than individual effects, average exposure estimates, which represent central tendencies, are compared against a derived intake of concern for populations. The IOC_{pop} incorporates a margin of safety with the intent of limiting exposures such that the probability of reaching "intervention" is significantly reduced for the large majority of children. No clear threshold for these effects has been identified in the epidemiological literature. Thus, even a very small exposure may carry a small degree of risk. No implicit judgement of acceptability is encoded because an acceptable daily intake is not suggested for lead. The question of concern has already been addressed through the regulatory policy of many environmental and state agencies which have adopted the CDC "threshold of concern". This level also coincides with the LOAEL identified here from which the IOC_{pop} is derived.

6.1 POPULATIONS AT RISK

Young children and the fetus represent the most vulnerable segments of the population, primarily because of the susceptibility of the developing nervous and other organ systems to toxic insult. Because the fetus is identified as a sensitive subgroup, pregnant women (as a surrogate for fetal exposure) also define a population for which risk should be minimized. A further consideration is that fetal exposure to lead, in most cases, may be dominated by maternal lead re-mobilized from bone lead stores. Therefore historical exposure for women of child-bearing age may be an important consideration. A lack of adequate biokinetic data prevents modelling of fetal exposures directly; this would require information on mobilization of bone lead stores and their transfer across the placenta. This dynamic process has not been quantified. Effects on blood pressure are the critical effect for adults, although the question of carcinogenicity is emerging and requires extensive research.

Until recently, misperceptions have persisted concerning lead poisoning in young children and these have influenced environmental and public health interventions. Childhood lead-poisoning was thought to be inextricably linked to old dilapidated housing and therefore limited to children living in old American inner city slums (Lin-Fu, 1992). While children in these areas are obviously in higher risk groups, excess exposure to lead affects children of all

socio-economic backgrounds. Until the 1980's, lead poisoning was viewed as a clinically symptomatic disease. Several studies have now demonstrated that many children encountering elevated lead exposure will be asymptomatic while a number of subclinical effects may still occur (See Chapter 2).

Given the greater sensitivity of the fetus and young children, potential risks associated with their exposures should form the emphasis of lead reduction and regulatory strategies. Because fetal and early childhood exposures are both periods of higher vulnerability to lead, no distinction should be made between the fetus, neonates, young children or adult groups (which include pregnant women and women of child-bearing age) in the development of preventative and reduction strategies for environmental lead exposure.

It is widely recognized that children living in the vicinity of point source emissions of lead are at higher risk of environmental lead exposure. This includes children living in the vicinity of secondary lead smelters or metal foundries, or areas contaminated with mine tailings. Children living in communities with historical or ongoing point-source emissions demonstrate higher blood lead levels than those in non-contaminated areas, although their blood leads are now notably lower over the last decade (e.g., South Riverdale area following abatement). These declines are not necessarily attributable to site-specific remedial measures. Whether or not the lead abatement activities are responsible for this decline as opposed to general declines in background exposure to lead, is currently the subject of an intensive study sponsored by the Ontario Ministry of Environment and the City of Toronto.

Lead paint in or on dwellings can be viewed as a recalcitrant hazard. Although many other exposures to lead have been significantly reduced, lead paint may have remained untouched for many years. While lead-containing paint has been recognized as a major problem in the United States, there has long been a perception that it is not a problem in Canada. It is concluded here that Ontario children living in such homes are at higher risk of potential lead exposure. Concentrations of lead in old paints range from 1100 to 330,000 ppm, based on samples of flaking external paint collected in 1990 (MOEE, unpublished data). The exposure scenarios presented in this document suggest possible intakes considerably greater than intake levels of concern. Therefore children living in such houses are at particular hazard to excess lead exposure through direct ingestion of paint particles or through ingestion of soil/dust contaminated with stripped or weathered paint. Particularly high exposures may occur in homes where renovation or remodelling is undertaken. The few reported cases of acute lead poisoning in Canada are usually associated with lead-based paint (Tenenbein, 1990). These occurrences may increase with the increase in young families purchasing and remodelling older homes (Lin-Fu, 1992).

Limited evidence suggests that native peoples may constitute a special population group at risk of higher exposure to lead. In the 1987 Northern Ontario Blood Lead Study, blood lead levels in the community of Moosonee (selected as the remote location for the survey) were comparable to urban levels; the mean blood lead level was 9.68 µg/dL. It was speculated that use of leaded gasoline and dietary characteristics, such as consumption of game

containing lead shot, may have contributed to this. Elevated blood lead levels have also been observed in Greenlandic Eskimos (Hansen, 1990) and again, food was believed to be the likely source. Greater use of canned foods in remote communities could be a dietary contributor to lead intake. Blood lead studies of several hundred children are now underway in 1992 in Moosenee and Moose Factory to more closely examine the blood lead status of native peoples (L. Smith, personal communication).

6.2 QUANTIFICATION OF ESTIMATED ONTARIO POPULATION RISKS

6.2.1 Young Children

The most direct and appropriate way to ascertain lead-related risk in a population is through the systemic survey of a large sample of children's blood lead levels throughout the province. Ongoing screening is not routinely carried out for the general population, except in high risk populations such as the South Riverdale community in Toronto. A provincially-based survey has not been conducted since the Northern Ontario Blood Lead Study of 1987. However, blood lead levels in Ontario are believed to compare very favourably with those in any other jurisdiction.

The Exposure Assessment suggests that typical exposures for children have declined by at least 50% since the mid-1980's. The decline is linked to decreased environmental inputs of lead and also to changes in the manner of food storage. Estimated total daily intakes for an average urban infant are currently estimated to be 1.12 $\mu\text{g/kg/day}$ for breast-fed infants (probably the majority of neonates today) and 2.85 $\mu\text{g/kg/day}$ for those fed cow's milk. Typical young children (0.5-4 years) are estimated to have total multimedia intakes of 2.87 $\mu\text{g/kg/day}$, or roughly 1.5 times the derived intake of concern for this population. Their level of dietary intake may have declined in recent years. If the projected value based on American dietary exposure figures is assumed (0.5 $\mu\text{g/kg/day}$), total daily exposures would be about 1.9 $\mu\text{g/kg/day}$, only slightly above the suggested intake of concern.

The typical chronic exposure from food, air, soil and drinking water is probably increased under specific scenarios, such as proximity to a point source, elevated drinking water levels of lead, and consumption of vegetables grown in lead-contaminated soils. Examination of these scenarios using average assumptions for each, suggests that total intakes are approximately equivalent (3.5-3.9 $\mu\text{g/kg/day}$). This is about 1.4 times the projected intake level for typical urban children. High soil ingestion rates in a child can lead to total daily intakes well above the levels associated with toxicity.

Chronic exposure may also be punctuated by intermittent acute exposures to consumer products. The latter are assessed under plausible scenarios of paint ingestion and consumption of fruit juices contaminated by lead glazes. The exposures are clearly many times higher than the derived intake of concern or any other quantitative measure of lead poisoning. Such exposures when they occur, may or may not be clinically recognized.

Unrecognized and untreated acute exposures would add significantly to the cumulative burden of chronic lead exposure.

The exposure estimates strongly suggest that, for typical urban children, the margin of safety between actual exposure and intakes associated with concern for potential neurobehavioural effects is small, if it exists at all. Under different higher exposure scenarios, total daily intakes may well exceed the intakes of concern. It is also important to note that although the total exposure for young children in Ontario has declined by at least 50% in the last decade, the blood lead level of concern for children has also declined by a roughly equivalent margin. Therefore, while actual levels of risk have declined substantially and may decline more, the degree of risk has not changed substantially. Rather, the previous risk level was underestimated by at least a factor of two. Further blood lead surveys of Ontario children will continue to provide the best picture of lead exposure in the 1990's.

Risk characterization also encompasses an estimation of the number of individuals in a population who are predicted to be at different levels of risk. As a PbB of 10 µg/dL has been associated with adverse effects, in the absence of a broad scale survey, estimates of numbers of children at higher risk (i.e. PbB > 10 µg/dL) are derived by extrapolating the results of the 1990 control survey of downtown Toronto children. The survey was conducted as part of the MOEE/City of Toronto Soil Lead Abatement Evaluation study (MOEE, unpublished data). These are considered the most representative data available. Of the 177 children (age <1-5 years) in the South Riverdale control area, seven children (4.0 %) had blood lead levels greater than 10 µg/dL; none was greater than 20 µg/dL. Highest mean levels were seen in children one year of age. In a sample of 245 children (age 3-6 years) from schools previously sampled in the 1984 Ontario Blood Lead Survey, eight children (3.3%) had blood leads above 10 µg/dL; only one child had a level greater than 20 µg/dL.

If the 1990 Toronto urban sample (432 children) is taken as representative of the blood lead distribution in urban areas, then about 4% of urban children in Ontario are estimated to have blood lead levels greater than 10 µg/dL. Data from the most recent population census are not yet available from Statistics Canada. Projections indicate a population of 564,900 children aged one to four years in Ontario in 1991 (Statistics Canada, 1992, personal communication). Approximately 151,000 children are less than one year of age. Based on 1986 census data, 80% of Ontario children live in urban areas, which would indicate approximately 452,000 urban children aged one to four years (Statistics Canada, 1987a, b). Applying the 4% value yields an estimated 18,000 Ontario children who may have blood lead levels greater than 10 µg/dL and are therefore at higher risk. This number may be somewhat larger (+ 8700) if children aged five and six years are included in the estimates.

Lead-based paints are identified as a potentially large source of exposure. It is difficult to estimate the extent of this hazard or to quantify the numbers of young children potentially exposed in Ontario. If one assumes that private dwellings built prior to 1970 may contain lead paints, this would represent 2,056,850 private dwellings in Ontario (Statistics Canada,

1987a). This question requires further research as to the prevalence of lead-based paint in Ontario homes and the numbers of young children inhabiting these homes.

6.2.2 Pregnancy and Infants

Quantifying the risks to the fetus is more difficult, given the uncertainties regarding the kinetics of lead distribution during pregnancy. Also, little is known about blood lead levels in female adult populations in Ontario. The projected number of live births in Ontario in 1987 was 134,617 (MTE, 1988). A 1989 study among 95 mother-infant pairs was conducted at Women's College Hospital in Toronto (Koren *et al.*, 1990). None of the randomly examined babies had blood lead levels of 10 $\mu\text{g}/\text{dL}$ or greater; 99% had levels less than 3 $\mu\text{g}/\text{dL}$. Compared to other cities like Boston and Port Pirie, Australia, the study suggests a lower level of risk in Toronto. Blood lead levels in mothers were consistently higher than those of newborns. This suggests that the degree of fetal lead exposure is generally smaller than that for young children. Maternal blood lead levels are highly correlated with fetal blood lead and the lead intake for adult women (0.81 $\mu\text{g}/\text{kg}/\text{day}$) is estimated to be considerably less than that for children on a body weight basis. On the other hand, the study was not designed to identify women at higher risk of lead exposure: women working or living in lead-polluted industrial areas or women occupationally exposed to lead.

In the control area of the 1990 Toronto study, a geometric mean blood lead level of 2.89 ($n=21$) was observed in infants less than one year old. It is therefore likely that exposures increase shortly after birth. The exposure estimate of 1.12 $\mu\text{g}/\text{kg}/\text{day}$ for breast-fed infants is less than one-half the intake level of concern. It would therefore appear that the risk of the environmental exposures of very young infants are notably smaller. Dietary components, particularly formula stored in cans with lead-soldered seams, may increase total exposures to undesirable levels.

6.3 MAGNITUDE OF RISK - HEALTH SIGNIFICANCE OF NEUROBEHAVIOURAL DEFICITS IN YOUNG CHILDREN

Of particular importance for deriving criteria and developing policy to protect public health are findings related to quantitative dose-response relationships for lead. As previously described this document, several prospective studies in child populations have provided valuable information on the magnitude of neurobehavioural deficits relative to increments in blood lead level. A pivotal question is: what are the consequences, both to the individual and to society, of these effects? For example, what is the public health significance of a two to eight point decline in the Bayley Mental Development Index (MDI) or of a change of three or four IQ points?

The toxicological significance of a four to eight point reduction in IQ or MDI score may at first appear unimportant. The standard deviation of the normal distribution of scores on the

Bayley MDI index is approximately 16 points. Therefore it could be argued that such an effect is not significant for an individual because it lies within the normal variability of the test method. Furthermore the effect is so subtle it may not be truly adverse. However, such seemingly small effects may be of considerable significance if viewed as an impact across the entire population.

It can be calculated statistically that a decline of five points in mean IQ score (assuming that distribution is maintained) results in a greater than two-fold increase in the percentage of children with an IQ below 70 (mentally retarded) and a decrease in the number of children in gifted categories. Needleman has predicted that a 6-point shift in the median score is associated with a four-fold increase in the risk of IQ below 80 (Needleman, 1989, 1990). This shift truncates the upper end of the curve with a reduction of children with IQ greater than 125 (superior function).

Similar analyses in shifts of IQ scores are being applied by the International Committee on Radiological Protection for regulatory purposes. The focus is on the effects of radiation exposure during prenatal development on IQ scores (ICRP, 1991).

For children below three years of age, for whom measures of intelligence are not available, similar potential downward shifts have been observed in scores such as MDI. An overall downward shift in MDI of four points would result in 50% more children scoring below 80 on these exams (Davis and Svendsgaard, 1987).

The benefits of avoiding such population risks could include reducing the potential demand for social program support, special education and facilities; and avoiding lost earnings for individuals. Although much more difficult to monetize, the impacts of reductions in the number of individuals in superior categories may be more far-reaching.

6.4 UNCERTAINTY ASSESSMENT

Relative to most other chemical risk assessments, the degree of confidence with the assessment of lead is high. The database associated with the critical effects is large and contains studies of high quality and strong analytical assessment. The conclusions are drawn from a studies of large numbers of children and thus conclusions are drawn directly from work on most sensitive receptor and the uncertainties associated with extrapolation from other species or subpopulations are avoided.

There are some uncertainties associated with the dose-response assessment for lead and the derivation of an intake of concern. These are considered relatively small as indicated by the small uncertainty factor applied in the derivation. One area of uncertainty surrounds the question of the lack of a discernible threshold for lead and the blood lead at which truly adverse effects begin to occur. The current discussion regarding the health significance of lead in terms of a population rather than an individual effect provides some uncertainty as

tolerable shift in population IQ has not been elicited. In deriving the intake of concern for child populations a principle uncertainty is in the selection of an intake/blood lead slope where there are variations (roughly 50%) in the relationships between oral intake and blood lead.

In the exposure assessment uncertainties arise in three general areas which apply to each media: 1.) analytical variation in levels of lead; 2.) variation in consumption patterns and representativeness of the estimate to the general population. For dietary exposure, a key uncertainty is that the consumption data for the Nutrition Canada survey are significantly dated (1972) and may not reflect the current diversity of populations and food consumption patterns or reflect new food trends (e.g fast foods). Further because the total diet survey data of 1985 precedes the phaseout of lead in the food canning industry, these levels may overestimate current levels in food. Also individual differences in food consumption for children and adults can vary by hundreds of grams per day. This introduces a moderate uncertainty in the dietary and thus total exposure estimates. The principle uncertainty in the soil/dust estimates are the soil ingestion assumptions for children and adults. A number of factors preclude strong empirical support for the soil ingestion value in children. No empirical evidence is available for adults. Therefore the estimates provided must be considered crude and a moderate degree of uncertainty associated with the estimates. For drinking water exposure, a conservative mean value was selected. As indicated with special scenarios some areas may have more plumbosolvent water supplies. Therefore although the mean may be conservative, individual water supplies may have much higher concentrations. The consumption figure utilized for children was 0.6 L and 1.5 for adults. This is also variable and typical values of up to 2 L have been utilized for adults. Overall, the drinking water intake may be quite variable, however the degree of uncertainty in the average intake estimate is considered relatively small.

Overall, there are moderately small areas of uncertainty in both the dose-response and exposure assessment areas as outlined. The overall confidence in this assessment comparable to most other contaminants is high. The estimates in the exposure calculations are also roughly supported by the observable trends in blood lead levels in various Ontario populations over the past decade. The degree of decline in actual blood leads roughly reflect the degree of decline as estimated by calculation, thereby increasing confidence in the estimation.

6.5 SOCIAL FACTORS INFLUENCING LEAD RISK

The impact of social factors on IQ and other mental performance measures have typically been examined in the epidemiological studies from which the current blood lead level of concern has been derived. In characterizing the risk of lead exposure on intelligence measures, it is important to note that the association of social indicators, such as social class and home environment, are always stronger than any lead/IQ association.

An important factor which may influence the overall risks for the Ontario population of children is that of socio-economic status. Several different studies of normal populations in have confirmed the relationship between lead measures and social indicators of disadvantage. Lack of suitable play areas, missed meals and low standards of family hygiene are all factors which may contribute to increased lead burden. Roughly one in six children in Ontario lived in poverty in 1990 (MCSS, 1992). It is estimated that between 1989 and 1990 the number of poor children in Ontario increased by 34% due largely to the onset of economic recession. This number is likely to rise in 1991 and 1992. The number of children in homeless families is also increasing. The living circumstances of these children may place them at increased risk of lead exposure.

6.6 SOME GUIDING CONSIDERATIONS FOR MULTIMEDIA STANDARDS DEVELOPMENT

Appendices A through D provide information on analytical methods, sources, emissions and environmental levels and transport which bear on the consideration of an appropriate standard. A comprehensive summary of international lead regulation is provided in Appendix I.

A number of general observations may be made on the question of lead regulation from a multimedia perspective. They reflect, in part, the initiatives and experience of credible agencies, the Ministry's own experience in using multimedia approaches for standards development and the outcomes of the present analysis. These considerations bear on the interface between scientific assessment and risk management and are provided as advice towards formulation of recommended multimedia guidelines.

- For the appropriate and effective development of health-based multimedia guidelines as well as strategies for exposure prevention and reduction, the Ministry should ensure that all program areas utilize a single consistent scientific approach and toxicological assessment. Standards development will provide an essential component in the overall strategy, but other companion approaches may be required, some of which may not necessarily fall into the regulatory purview of the Ministry. For example guidelines for allowable levels of lead in paint and for lead-based paint removal would not be within the MOEE mandate. However, the multimedia assessment can provide useful information which may influence decisions in these related areas.

- The most appropriate focus for regulatory action is prevention of potential neurobehavioural effects in young children and infants. Assessment of risk should be based on these effects. Measures protective of children should reasonably ensure that environmental lead intakes and resultant blood lead levels remain low enough to limit risk for older age groups. Quantitative

assessment of lead as a carcinogen is not warranted given the large uncertainties in extrapolating from animal data.

- The multimedia exposure profile for young children, as opposed to other age groups, is the most appropriate exposure framework for decisions regarding the allocation of exposure to specific pathways. It is necessary to consider all related lead guidelines together, particularly in view of the lack of a significant margin between estimated exposures and the suggested intake of concern.
- The goal of all lead exposure prevention and actions should be to reduce children's blood lead levels below 10 µg/dL, the recently recognized blood lead level of concern.
- Specific health-based guidelines should rely on the derived intake of concern for child populations (IOC_{pop}) of 1.85 µg/kg/day. This value accommodates the population-based significance of the health effects and minimizes the predicted number of children with individual blood lead levels of concern.

6.6.1 Apportionment of Exposure

In setting guidelines for lead, risk management factors, such as analytical detection limits, technical feasibility of control, and socio-economic factors will also need to be considered. Allocation of exposure among media, is a risk management exercise which is influenced by these factors. It is generally based initially on the relative contribution of exposures as indicated by the exposure assessment and modified thereafter. The following examples of allocations are not recommendations. They are intended to outline the key information as well as to provide examples of possible "sets" of media-specific intake limits.

Traditionally, in developing health-based guidelines based on multimedia principles, the relative contribution of various pathways from typical or normal daily exposure are applied to a tolerable intake or reference dose. This is to ensure that total exposure does not exceed the latter value. It must be recognized that no threshold or safe value has been identified for lead. However, an intake of concern, which should keep the majority of children below the individual blood lead level of concern, can serve as a guiding value.

As a guiding framework and starting point for such decisions, the exposure profile(s) for children should be used. Consideration of the exposure information provides various options including the following:

Option 1: If the relative contributions are applied directly to the IOC_{pop} , this would translate into media-specific doses in Table 6.1.

TABLE 6.1 MEDIA-SPECIFIC DOSES BASED ON IOC_{pop}

MEDIA OR SUBSTRATE	RELATIVE CONTRIBUTION (%)	CORRESPONDING INTAKE (µg/kg/day)
Food	48	0.88
Drinking water	8	0.15
Air	<1	0.02
Soil/dust	43	0.79

Option 2: If the food intake estimates are adjusted downward on the basis of projection to 1992 as opposed to using actual 1985 food data, this would translate into the media-specific doses for a typical urban child (Table 6.2).

TABLE 6.2 MEDIA-SPECIFIC DOSES USING PROJECTED 1993 FOOD DATA

MEDIA OR SUBSTRATE	RELATIVE CONTRIBUTION (%)	CORRESPONDING INTAKE (µg/kg/day)
Food	24	0.44
Drinking water	12	0.20
Air	<1	0.02
Soil/dust	64	1.18

Other approaches could also consider allocation at potential sources (e.g. the input of air lead to soil lead) as opposed to allocation at the receptor.

SUMMARY

No clear threshold for the neurobehavioural effects of lead has been identified in the epidemiological literature. Thus, even a very small exposure may carry a small degree of risk.

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- Average exposure estimates, which represent central tendencies, are compared against a derived intake of concern for populations. The IOC_{pop} incorporates a margin of safety with the "preventative" intent of limiting exposures such that the probability of reaching "intervention" is significantly reduced for the large majority of children.
 - Young children and the fetus represent the most vulnerable segments of the population, primarily because of the susceptibility of the developing nervous and other organ systems to toxic insult. Because the fetus is identified as a sensitive subgroup, pregnant women (as a surrogate for fetal exposure) also define a population for which risk should be minimized. A lack of adequate biokinetic data prevents modelling of fetal exposures directly; the degree of fetal lead exposure is generally smaller than that for young children.
 - Because fetal and early childhood exposures are both periods of higher vulnerability to lead, no distinction should be made between the fetus, neonates, young children or adult groups (which include pregnant women and women of child-bearing age) in the development of preventative and reduction strategies for environmental lead exposure.
 - It is widely recognized that children living in the vicinity of point source emissions of lead are at higher risk of environmental lead exposure.
 - Children living in homes containing lead paint who may be exposed to this hazard are another higher risk group. Modelled intakes are considerably greater than intake levels of concern. Therefore children living in such houses are at particular hazard to excess lead exposure through direct ingestion of paint particles or through ingestion of soil/dust contaminated with stripped or weathered paint. Particularly high exposures may occur in homes where renovation or remodelling is undertaken. Lead poisoning occurrences may increase with the increase in young families purchasing and remodelling older homes.
 - Limited evidence suggests that native peoples may constitute a special population group at risk of higher exposure to lead.
 - The modelled exposures suggests that typical exposures for children have declined by at least 50% since the early 1980's. Typical young children (0.5-4 years) are estimated to have total multimedia intakes of $2.87 \mu\text{g/kg/day}$, or roughly 1.5 times the derived intake of concern for this population of $1.85 \mu\text{g/kg/day}$. Their level of dietary intake may have significantly declined in recent years. If the projected value based on American dietary exposure figures is assumed ($0.5 \mu\text{g/kg/day}$), total daily exposures would be about $1.9 \mu\text{g/kg/day}$, or just very slightly above the suggested intake of concern. Breast fed neonates are estimate to have total intakes below the IOC_{pop} whereas those fed canned or cow's milk may have total intakes at or above the IOC_{pop} .
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The exposure estimates strongly suggest that, for typical urban children, the margin of safety between actual exposure and intakes associated with concern for potential neurobehavioural effects is small, if it exists at all. Under different higher exposure scenarios, total daily intakes may well exceed the intakes of concern. It is also important to note that although the total exposure for young children in Ontario has declined by at least 50% in the last decade, the blood lead level of concern for children has also declined by a roughly equivalent margin. Therefore, while actual levels of risk have declined substantially and may decline more, the degree of risk has not changed substantially. Rather, the previous risk level was underestimated by at least a factor of two. Further blood lead surveys of Ontario children will continue to provide the best picture of lead exposure in the 1990's.

It is roughly estimated that potentially 18,000 Ontario children may have blood lead levels greater than 10 µg/dL and are therefore at higher individual risk.

It is difficult to estimate the extent of the lead in paint hazard or to quantify the numbers of young children potentially exposed in Ontario. If one assumes that private dwellings built prior to 1970 may contain lead paints, this would represent 2,056,850 private dwellings in Ontario. Further study would be required to clarify this question.

The apparently subtle neurobehavioural effects of lead may be of considerable significance if viewed as an impact across the entire population. It can be calculated statistically that a decline of five points in mean IQ score (assuming that distribution is maintained) results in a greater than two-fold increase in the percentage of children with an IQ below 70 (mentally retarded) and a decrease in the number of children in gifted categories.

An important factor which may influence the overall risks for the Ontario population of children is that of socio-economic status. The association of social indicators, such as social class and home environment, are always stronger than any lead/IQ association.

Roughly one in six children in Ontario live in poverty and the number of children in homeless families is also increasing. The living circumstances of these children may place them at increased risk of lead exposure.

Overall, there are moderately small areas of uncertainty in both the dose-response and exposure assessment areas as outlined. The overall confidence in this assessment comparable to most other contaminants is high.

A number of basic considerations for the development of multimedia lead guidelines are outlined as well as some options for allocation of the intake of concern for populations.

REFERENCES

- Ades, A.E., and Kazantzis, G. (1988) Lung cancer in a non-ferrous smelter: The role of cadmium. *Br. J. Ind. Med.* 45: 435-442.
- Agency for Toxic Substances and Disease Registry, ATSDR (1988). The nature and extent of lead poisoning in children in the United States: A report to Congress. Atlanta: Department of Health and Human Services, 1988.
- Agency for Toxic Substances and Disease Registry, ATSDR (1993) Toxicological Profile for Lead. Updated.
- Alessio, L., Foa, V. (1982) Human biological monitoring of industrial chemicals 4: Inorganic Lead. *Comm. Eur Communities Eur Rep.* 1-57.
- Alexander, F.W.; Delves, H.T. (1981) Blood lead levels during pregnancy. *Int. Arch. Occup. Environ. Health* 48: 35-39.
- Alexander, F.W., Delves, H.T.; Clayton, B.E. (1993). The uptake and excretion by children of lead and other contaminants. In: Environmental health aspects of lead proceedings, international symposium Amsterdam Commission of the European Communities. pp. 319-331.
- Alomran, A.H. and Shleamoon, M.N. (1988) The influence of chronic lead exposure on lymphocyte proliferative response and immunoglobulin levels in storage battery worker. *J. Biol. Sci. Res.* 19: 575-585.
- Alvares, A.P., Kapelner, S., Sassa, S. *et al.* (1975) Drug metabolism in normal children, lead-poisoned children and normal adults. *Clin. Pharmacol Ther.* 17: 179-183.
- American Academy of Pediatrics (1987) Statement on childhood lead poisoning. Committee on Environmental Hazards/Committee on Accident and Poison Prevention. *Pediatrics* 79: 457-462.
- Angle, C.R.; McIntire, M.S. (1979) Environmental lead and children: The Omaha study. *J. Toxicol. Environ. Health* 5: 855-870.
- Angle, C.R., McIntire, M.S., Swanson, M.S. *et al.* (1982) Erythrocyte nucleotides in children-increased blood lead and cytidine triphosphate. *Pediatr. Res.* 16: 331-334.
- Arati, S., Honma, T., Yanagihara, S. *et al.* (1980) Recovery of slowed nerve conduction velocity in lead-exposed workers. *Int. Arch. Occup. Environ. Health* 46: 151-157.
- Azar, A.; Trochimowicz, H.J. and Maxfield, M.E. (1973) as cited in USEPA (1989c).
-

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- Baghurst, P.A., Tong, S.L., McMichael, A.J., Robertson, E.F., Wigg, N.R., Vimpani, G.V. (1992a) Determinants of blood lead concentrations to age 5 years in a birth cohort study of children living in the lead smelting city of Port Pirie and surrounding areas. *Arch. Environ. Health* 47(3): 203-210.
- Baghurst, P.A., McMichael, A.J., Wigg, N.R., Vimpani, G.V., Robertson, E.F., Roberts, R.J., Tong, S.L. (1992b). Environmental exposure to lead and children's intelligence at the age of seven years: The Port Pirie cohort study. *N. Engl. J. Med.* 327 (18): 1279-1284.
- Baker, E.L., Landrigan, P.J., et al. (1979) Occupational lead poisoning in the United States: clinical and biochemical findings related to blood lead levels. *Br. J. Ind. Med.* 36:314-322.
- Baker, E.L., Goyer, R.A., et al. (1980) Occupational lead exposure, nephropathy and renal cancer. *Am. J. Ind. Med.* 1:139-148.
- Baker, E.L., Feldman, R.G., et al. (1984) Occupational lead neurotoxicity: A behavioural and electrophysiological evaluation. Study design and year one results. *Br. J. Ind. Med.* 41:352-361.
- Bayley, N. (1969) Bayley Scales of Infant Development. Psychological Corp., New York.
- Barltrop, D. (1969) Transfer of lead to the human foetus. In: Mineral Metabolism in Paediatrics. Edited by D. Barltrop and W.L. Burland. Publ. by Blackwell Scientific Publications, Oxford, p.135.
- Barltrop, D.; Strehlow, C.D.; Thornton, I.; Webb, J.S. (1974) Significance of high soil concentrations for childhood lead burdens. *Environ. Health Perspect.* 7: 75-82.
- Barltrop, D., Thornton, I., Strehlow, C.D., Webb, J.S. (1975) Absorption of lead from soil and dust. *Postgraduate Medical Journal* 51, 801-804.
- Barltrop, D.; Meek, F. (1979) Effect of particle size on lead absorption from the gut. *Arch. Environ. Health* 34: 280-285.
- Beck, B.D., (1992) An update on exposure and effects of lead. *Fundamental and Applied Toxicology* 18: 1-16.
- Bellinger, D.; Leviton, A.; Rabinowitz, M.; Needleman, H.; Waternaux, C. (1986) Correlates of low-level lead exposure in urban children at 2 years of age. *Pediatrics* 77: 826-833.
- Bellinger, D.C., Needleman, H.L., Leviton, A., Waternaux, C., Rabinowitz, M.B., and Nichols, M.L. (1984) Early sensory-motor development and prenatal exposure to lead. *Neurobehav. Toxicol. Teratol.*, 6: 387.
-

-
- Bellinger, D., Leviton, A., Waternaux, C., Needleman, H., Rabinowitz, M. (1987) Longitudinal analyses of prenatal and postnatal lead exposure and early cognitive development. *N. Engl. J. Med.* 316: 1037-43.
- Bellinger, D.; Leviton, A.; Waternaux, C.; Needleman, H.; Rabinowitz, M. (1989a) Low-level lead exposure and early development in socioeconomically advantaged urban infants. In: Smith, M.A.; Grant, L.D.; Sors, A.I., eds. Lead exposure and child development: An international assessment [international workshop on effects of lead exposure on neurobehavioural development]; September 1986; Edinburgh, United Kingdom. Lancaster, United Kingdom; Kluwer Academic Publishers; pp. 345-356.
- Bellinger, D.; Leviton, A.; Sloman, J. (1989b) Antecedents and correlates of improved cognitive performance in children exposed in utero to low levels of lead. Presented at: Conference on advances in lead research: Implications for environmental research. Research Triangle Park, NC: National Institute of Environmental Health Sciences, January.
- Bellinger, D., Sloman, J., Leviton, A., Rabinowitz, M., Needleman, H., Waternaux, C. (1991) Low-level exposure and children's cognitive function in the preschool years. *Pediatrics* 87:219-27.
- Betts, P.R., Astley, R., Taine, D.N. (1973) Lead intoxication in children in Birmingham. *Br. Med. J.* 1: 402-406.
- Binder, S.; Sokal, D.; Maughan, D. (1986) Estimating soil ingestion: The use of tracer elements in estimating the amount of soil ingested by young children. *Arch. Env. Health* 41: 341-345.
- Blake, K.C.H., Barbezat, G.O., Mann, M. (1983a) Effect of dietary constituents on the gastrointestinal absorption of lead-²⁰³ in man. *Environ. Res.* 30(1): 182-187.
- Bornschein, R.L., Succop, P.A., Krafft, K.M., Clark, C.S., Peace, B., Hammond, P.B. (1986) Exterior surface dust lead, interior house dust lead and childhood lead exposure in an urban environment. In: Hemphill, D.D., ed. Trace substances in environmental health. Columbia (MO): University of Missouri, 322-32.
- Bornschein, R.L.; Grote, J.; Mitchell, T.; Succop, P.A.; Dietrich, K.N.; Krafft, K.M.; Hammond, P.B. (1989) Effects of prenatal lead exposure on infant size at birth. In: Smith, M.A.; Grant, L.D.; Sors, A.I., eds. Lead exposure and child development: An international assessment [international workshop on effects of lead exposure on neurobehavioural development]; September 1986; Edinburgh, United Kingdom. Lancaster, United Kingdom: Kluwer Academic Publishers; pp. 307-319.
-

-
- Brunekeerf, B.; Veenstra, S.J.; Biersteker, K.; Boleij, J.S.M: (1981) The Arnhem lead study: 1. lead uptake by 1- to 3-year-old children living in the vicinity of a secondary lead smelter in Arnhem, The Netherlands. *Environ. Res.* 25: 441-448.
- Bull, R.S., Lutkenboff, S.D., McCaarty, G.E. *et al.* (1979) Delays in the postnatal increase of cerebral cytochrome concentrations in lead exposed rats. *Neuropharmacology* 18: 83-92.
- Campara, P., D'Andrea, F., Micciolo, R., Savonitto, C., Tansella, M. and Zimmermann-Tansella, C. (1984) Psychological performance of workers with blood-lead concentration below the current threshold limit value. *Int. Arch. Occup. Environ. Health*, 53: 233.
- Campbell, B.C., Beattie, A.D., Moore, M.R., Goldberg, A. and Reid, A.G. (1977) Renal insufficiency associated with excessive lead exposure. *Br. Med. J.* (i): 482.
- Centers for Disease Control (CDC) (1985) Preventing lead poisoning in young children. U.S. Department of Health and Human Services. January, 1985.
- Centers for Disease Control, CDC (1991) Preventing lead poisoning in young children. A Statement by the CDC-October 1991. U. S. Department of Health and Human Services
- Chamberlain, A.C.; Heard, M.J.; Little, P.; Newton, D. Wells, A.C.; Wiffen, R.D. (1978) Investigation into lead from motor vehicles. Harwell, United Kingdom: United Kingdom Atomic Energy Authority; Report No. AERE-R9198.
- Charney, E.; Kessler, R.; Farfel, M.; Jackson, D. (1983) Childhood lead poisoning: a controlled trial of the effect of dust-control measures on blood lead levels. *N. Engl. J. Med.* 309: 1089-1093.
- Chisolm, J.J. (1962) Aminoaciduria as a manifestation of renal tubular injury in lead intoxication and a comparison with patterns of aminoaciduria seen in other diseases. *J. Pediatr.* 60: 1-17.
- Chisolm, J.J. (1965) Chronic lead intoxication in children. *Dev. Med Child Neurol.* 7: 529-536.
- Chisolm, J.J., Mellits, E. and Barrett, M.B. (1976) Interrelationships among blood lead concentrations, quantitative daily ALA-V and urinary lead output following calcium EDTA. In: Nordberg, G.F. ed. Proceedings of the third meeting of the subcommittee of the toxicology of metals.
- Clausing, P.; Brunekeerf, B.; van Wijnen, J.H. (1987) A method for estimating soil ingestion by children. *Int. Arch. Occup. Environ. Health* 59: 73-82.
-

-
- Cohen, A.F.; Cohen, B.L. (1980) Protection from being indoors against inhalation of suspended particulate matter of outdoor origin. *Atmos. Environ.* 14: 183-184.
- Cohen, J. (1988) as cited in USEPA 1989a. Revision to dietary lead estimates for case study exposure analyses. Memorandum to the files, U.S. EPA, Office of Air Quality Planning and Standards, Ambient Standards Branch, RTP, September 9, 1988.
- Cools, A., Salle H.J., Verbeck, M.M. and Ziehuls (1976) Biochemical response of male volunteers ingesting inorganic lead for 49 days. *Int Arch. Occup. Environ. Health.* 38, 129-139.
- Cooper, W.C. (1976) Cancer mortality patterns in the lead industry. *Ann. N.Y. Acad. Sci.* 271: 250-259.
- Cooper, W.C., Womg. O. and Kheifets, L. (1985) Mortality among employees of lead batter plants and lead-producing plants, 1947-1980. *Scand. J. Work Environ. Health*, 11: 331-345.
- Cory-Schelta, D.A., Bissen, S.T., Young, A.M., and Thompson, T. (1989) Chronic post-weaning lead exposure and response duration performance. *Tox. Appl. Pharmacol.* 60:78-84.
- Czuba, M. and Hutchinson, T. C. (1980) Copper and lead levels in crops and soils of the Holland Marsh area - Ontario. *J. Environ. Qual.* 9(4): 566-574.
- Dabeka, R. (1986) Dietary lead intakes in health effects of lead. Royal Society of Canada. Commission on Lead in the Environment.
- Dabeka, R.W., Arthur D. McKenzie and Gladys M.A. Lacroix (1987) Dietary intake of lead, arsenic and fluoride by Canadian adults: A 24-hour duplicate diet study. *Food Additives and Contaminants*, 4: 89-102.
- Dabeka, R.W. and Mackenzie, A.D. (1987a) Survey of lead, cadmium, cobalt and nickel in infant formula and evaporated milks and estimates of dietary intakes of elements by infants 0-12 months. *Sci. Total Env.* 89: 279-289.
- Dabeka, R. W. and Mackenzie, A.D. (1987b) Lead, cadmium and fluoride levels in market milk and infant formulas in Canada. *J. Assoc. Off. Anal. Chem.* 70:754-757.
- Dabeka, R.W. and McKenzie, A.D. (1991) Total diet study of lead and cadmium in food composites. Preliminary investigations. Draft submitted publication.
- Dacre, J.C.; Ter Haar, G.L. (1977) Lead levels in tissues from rats fed soils containing lead. *Arch. Environ. Contam. Toxicol.* 6: 111-119
-

-
- Davis, J.M., Svendsgaard, D.J. (1987) Lead and child development. *Nature* 329:297-300.
- de la Burde, B. and Choate, M.S. Does early asymptomatic lead exposure in children have latent sequelae? *J. Pediatrics* 81: 1088-1091.
- Deknudt, G., Manuel, Y. and Gerber, G.B. (1977) Chromosomal aberrations in workers professionally exposed to lead. *J. Toxicol. Environ. Health* 3: 885-891.
- Dietrich, K.N.; Krafft, K.M.; Bier, M.; Succop, P.A.; Berger, O.; Bornschein, R.L. (1986) Early effects of fetal lead exposure: Neurobehavioural findings at 6 months. *Int. J. Biosoc. Res.* 8:151-168.
- Dietrich, K.N., Krafft, K.M., Bornschein, R.L., Hammond, P.B., Berger, O., Succop, P.A., Bier, M. (1987a) Low-level fetal lead exposure effect on neurobehavioral development in early infancy. *Pediatrics* 80:721-30.
- Dietrich, K.N.; Krafft, K.M.; Shukla, R.; Bornschein, R.L.; Succop, P.A. (1987b) The neurobehavioural effects of early lead exposure. In: Schroeder, S.R., ed. Toxic substances and mental retardation: neurobehavioural toxicology and teratology. Washington, D.C.: American Association on Mental Deficiency: pp. 71-95. (Begab, M.J., ed. Monographs of the American Association on Mental Deficiency: no. 8).
- Dietrich, K.N.; Krafft, K.M.; Bier, M.; Berger, O.; Succop, P.A.; Bornschein, R.L. (1989a) Neurobehavioural effects of foetal lead exposure: the first year of life. In: Smith, M.A.; Grant, L.D.; Sors, A.I., eds. Lead exposure and child development: An international assessment [international workshop on effects of lead exposure on neurobehavioural development]: September, 1986; Edinburgh, United Kingdom. Lancaster, United Kingdom: Kluwer Academic Publishers; pp. 320-331.
- Dietrich, K.N.; Succop, P.A.; Bornschein, R.L.; Krafft, K.M.; Berger, O.; Hammond, P.B.; Buncher, C.R. (1989b) Lead exposure and neurobehavioural development in later infancy. Presented at: Conference on advances in lead research: Implications for environmental research. Research Triangle Park, NC: National Institute of Environmental Health Services; January.
- Dietrich, K.N., Succop, P.A., Berger, O., Hammond, P., Bornschein, R.L. (1991) Lead exposure and cognitive development of urban preschool children: The Cincinnati lead study cohort at age 4 years. *Neurotoxicology and Teratology* 13:203-11.
- Drill, S.; Konz, J.; Mahar, H.; Morse, M. (1979) The environmental lead problem: An assessment of lead in drinking water from a multi-media perspective. Washington, D.C.: U.S. Environmental Protection Agency; EPA report no. EPA-570/9-79-003. Available from NTIS, Springfield, VA; PB 296556.
-

-
- Duggan, M.J.; Inskip, M.J. (1985) Childhood exposure to lead in surface dust and soil: a community health problem. *Public Health Rev.* 13: 1-54.
- Duncan, C., Kusiak, R.A., O'Heany, J., Smith, L.F. and Spielberg, L. (1985) "Blood Lead and Associated Risk Factors in Ontario Children, 1984, Report for Ontario Ministry of Health, Ministry of Labour and Ministry of the Environment.
- Elwood, P.C., Daver-Smith, G., Oldman, P. *et al.* (1988) Two Welsh surveys of blood lead and blood pressure. *Environ. Health Perspect.* 78: 119-121.
- Ernhart, C., Landa, B. and Schell, N.B. (1981) Subclinical levels of lead and development deficit - A multivariate follow-up reassessment. *Pediatrics*, 67: 911.
- Ernhart, C.B., Landa, B. and Wolf, A.W. (1985) Subclinical lead level and developmental deficits: Reanalysis of data. *J. Learn. Disabil.* 18: 475-479.
- Ernhart, C., Wolf, A., Kennard, M., *et al.* (1986) Intrauterine exposure to low levels of lead: The status of the neonate. *Arch. Environ. Health* 41: 287-291.
- Ernhart, C.B., Morrow-Tlucate, M., Marler, M. and Wolf, A. (1987) Low level lead exposure in the prenatal and early preschool periods: Early preschool development. *Neurotoxicol. Teratol.* 9: 259-270.
- Ernhart, C.B. and Morrow-Tlucate, M. (1989) Low-level lead exposure and intelligence in the early pre-school years. In: Lead exposure and child development: An international assessment. pp. 469-475.
- Ernhart, C.B., Brittenham, G., Marten, M. and Sokol, R. (1989) Lead-related birth defects: some methodological issues. In: Lead Exposure and Child Development: An international assessment. pp. 357-379.
- Ewers, U., Stiller-Winkler, R., Idil, H., (1982) Serum immunoglobulin complement C3' and salivary IgA level in lead worker. *Environ. Res.* 29: 351-357.
- Facchetti, S and Geiss, R. (1985) Isotopic lead experiment - An update. Presented at: Lead Environmental Health: The current issue, Durham, N.C., Duke University Medical Center.
- Fergusson, D.M.; Fergusson, J.E.; Horwood, L.J.; Kinzett, N.G. (1988a) A longitudinal study of dentine lead levels, intelligence, school performance, and behaviour. Part II: Dentine lead and cognitive ability. *J. Child Psych. Psychiat.* 29:793-809.
-

-
- Fergusson, D.M.; Fergusson, J.E.; Horwood, L.J.; Kinzett, N.G. (1988b) A longitudinal study of dentine lead levels, intelligence, school performance and behaviour: Part III. Dentine lead levels and attention/activity. *J. Child Psychol. Psychiatry Allied Discip.* 29: 811-824.
- Flegel, A.R. (1985) Lead in a pelagic food chain. Pages 83-90 In: J. Salanki (ed.). Heavy metals in water organisms. Symposia Biologica Hungarica, Vol. 29. Akademiai Kiado, Budapest, Hungary.
- Flegel, A.R.; Smith, D.R.; Elias, R.W. (1988) Lead contamination in food. In: Nriagu, J.O.; Simmons, M.S., eds. Environmental Food Contamination; Advances in Environmental Science and Technology. J. Wiley, New York.
- Frank, R, Ishida, K and Suda, P (1985) Metals in agricultural soils of Ontario. *Canadian Journal of Soil Science*, 56:181-196.
- Frank, R, Braun, H., Suda, P., Ripley, B.D *et al.* (1987) Pesticide residues and metal contents in flue-cured tobacco and tobacco soils of southern Ontario, Canada, 1980-85. *Tobacco Sci.* 31: 40-45.
- Fulton, M., Raab, G.; Thomson, G.; Laxsen, D.; Hunter, R.; Hepburn, W. (1987) Influence of blood lead on the ability and attainment of children in Edinburgh. *Lancet* (8544): 1221-1226.
- Gartrell, M. Craun, J. Podebrac, D and Gunderson, E. (1986) Pesticide, selected elements and other chemicals in adult total diet samples, October 1980- March 1982. *J. Assoc. Off. Anal. Chem.* 69:146-160.
- Gartside, P.S. (1986) The relationship between blood lead levels and blood-pressure and its cardiovascular risk implications. [Letter]. *Amer. J. Epidemiol.*, 124: 864.
- Gerhardson, L., Lundstrom, N.G., Nordberg, G. and Wall, S. (1986) Mortality and lead exposure: A retrospective cohort study of Swedish smelter workers. *Br. J. Ind. Med.* 43: 707-712.
- Gilbert, S.G. and Rice, D.C. (1987) Low-level lifetime lead exposure produces behavioral toxicity (Spatial Discrimination Reversal) in adult monkeys. *Toxicol. Appl. Pharmacol.*, 91: 484.
- Goldberg, A.M., Meredith, P.A., Millers, *et al.* (1978) Hepatic drug metabolism and heme biosynthesis in lead in lead-poisoned rats. *Br. J. Pharmacol.* 62: 529-536.
-

-
- Goss, Gilroy and Associates (1989) Blood lead concentrations and associated risk factors in a sample of northern ontario children, 1987. Prepared for the Ontario Ministry of Health and Ontario Ministry of the Environment.
- Goyer, R.A. (1978) Calcium and lead interactions: Some new insights. *J. Lab. Clin. Med.* 91: 245-251.
- Goyer, R.A. (1985) Renal changes associated with lead exposures. In: Dietary and environmental lead: Human health effects. New York, Ny: Elviesier, pp. 315-338.
- Goyer, R.A. (1990) Transplacental transport of lead. Conference on advances in lead research: Implications for environmental health, Research Triangle Park, North Carolina, USA. *Environ. Health Perspect.* 89: 101-106.
- Graham, H.T. Data from distribution systems study, 1981, 1983, 1985-1991. Water Resources Branch, Ontario Ministry of the Environment. Personal communication.
- Graham, H.T. Ontario Lead Consumption Study using a Composite Sampler, 1988. Water Resources Branch, Ontario Ministry of the Environment. Personal Communication, Sept 1989.
- Grandjean, P. and Lintrup, J. (1978) Erythrocyte-Zn-protoporphyrin as an indicator of lead exposure. *Scand. J. Clin. Lab. Invest.*, 38: 669.
- Granick, J.L., Sassa, S., Granick, S., Levere, R.D. and Kappas, A. (1973) Studies in lead poisoning. II. Correlation between the ratio of activated to inactivated δ -aminolevulinic acid dehydratase of whole blood and the blood lead level. *Biochem. Med.*, 8: 149.
- Grant, L.D. and Davis, J.M. (1989) Effects of low level lead exposure on paediatric neurobehavioral development: current findings and future directions. In: Lead exposure and child development. An international assessment. M.A. Smith, L.D. Grant and A.I. Sors, eds. Kluwer academic, Boston pp. 49-118.
- Griffen, T., Coulston, F., Goldberg, L. et al. (1975) Clinical studies of men continuously exposed to airborne particulate lead. In: Griffen, T.B., Knelson, J.G, ed. Lead. Georg Thieme Publisher, 221-240.
- Gross, S.B. (1979) Oral and inhalation lead exposures in human subjects (Kehoe Balance experiments) New York, NY: Lead Industries Association.
- Gunderson, E (1991) personal communication. United States Food and Drug Administration, Washington.
-

-
- Haenninen, H., Mantere, P., Hernberg, S. *et al.* (1979) Subjective symptoms in low level exposure to lead. *Neurotoxicology* 1: 333-347.
- Hansen, J.C. (1990) Human exposure to metals through consumption of marine foods: A case study of exceptionally high intake among greenlanders.
- Harlan, W.R., Landis, J.R., Schmouder, R.L. *et al.* (1985) Blood and blood pressure relationships in the adolescent and adult U.S. population. *JAMA* 253: 530-534
- Harley, N.H., Kneip, T.H. (1985) An integrated metabolic model for lead in humans of all ages. Final report to the U.S. EPA, Contract No. B44899 with New York University School of Medicine, Dept. of Environmental Medicine, January 30, 1985.
- Harvey, P.G. (1986) Lead and children's intelligence test performance: A critical evaluative review of recent studies. *Rev. Environ. Health*, 6: 119.
- Harvey, P.G., Hamlin, M.W., Kumar, R.; Delves, H.T. (1984) Blood lead, behaviour and intelligence test performance in preschool children. *Sci. Total Environ.* 40: 45-60.
- Harvey, P.G., Hamlin, M.W., Kumar, R., Morgan, J., Spurgeon, A. and Delves, T. (1989) The Birmingham blood lead studies. In: Lead exposure and child development: An international assessment. M.A. Smith, L.D. Grant and A.I. Sors, eds. Kluwer Academic, Boston. pp. 201-211.
- Hass, G.M., McDonald, J.H., Oyasu, R. *et al.* (1965) as cited in U.S. EPA (1989c).
- Hatzakis, A.; Kokkevi, A.; Katsouyanni, K.; Maravelias, K.; Salaminios, F.; Kalandidi, A.; Koutselinis, A.; Stefanis, K.; Trichopoulos, D. (1987) Psychometric intelligence and attentional performance deficits in lead-exposed children. In: Lindberg, S.E.; Hutchinson, T.C., eds. International conference: Heavy metals in the environment, v. 1: September: New Orleans, LA. Edinburgh, United Kingdom: CEP Consultants, Ltd.; pp. 204-209.
- Hawk, B.A.; Schroeder, S.R.; Robinson, G.; Otto, D.; Mushak, P.; Kleinbaum, D.; Dawson, G. (1986) Relation of lead and social factors to IQ of low-SES children: A partial replication. *Am. J. Ment. Def.* 91: 178-83.
- HWC (Health and Welfare Canada) (1987). Draft. Supporting Documentation for a Revised Drinking Water Objective for Lead
- HWC (Health and Welfare) (1988) Draft Reference Values for Canadians. Environmental Health Protectorate.
-

-
- HWC (Health and Welfare Canada) (1990a) Present patterns and trends in infant feeding in Canada, Health and Welfare Canada, Ottawa.
- HWC (Health and Welfare Canada) (1990b) Revised Draft. Supporting Documentation for a Revised Drinking Water Objective for Lead. Federal/Provincial Subcommittee on Drinking Water Criteria
- Heard, M.J., Chamberlain, A.C. (1984) Uptake of Pb by human skeleton and comparative metabolism of lead and alkaline earth elements. *Health Physics* 47: 857-862
- Herber, R. (1980) Estimation of blood lead values from blood porphyrin and urinary delta-aminolevulinic acid levels in workers. *Int. Arch. Occup. Environ. Health*, 45: 169.
- Hernberg, S. and Nikkanen, J. (1970) Enzyme inhibition by lead under normal urban conditions. *Lancet* (i): 63.
- Hiasi, Y., Oshima, M., Kitahori, Y. Fujita, J., Yuasa, T and Myashiro, H. (1983) Basic lead acetate: Promoting effects of the development of renal tubular cell tumours in rats treated with N-ethyl-N-hydroxyethylnitrosamine. *J. Natl. Cancer. Inst.* 70:761-765.
- HMSO (1983) United Kingdom Royal Commission on Environmental Pollution. Ninth Report. Lead in the Environment. HMSO, London, April 1983.
- Hryhorczuk, D.O., Rabinowitz, M.B. *et al.* (1985) Elimination kinetics of blood lead in workers with chronic lead intoxication. *Am. J. Ind. Med.* 8: 33-42.
- Huston, B (1990) Personal Communication. Foods Directorate, Health and Welfare Canada.
- Hunt, T.J., Hepner, R. and Seaton, K.W. (1982) Childhood lead poisoning and inadequate child care. *Am. J. Dis. Child.*, 136: 538.
- ICRP (International Committee on Radiological Protection) (1975) Report of the Task Group on Reference Man: Report #23. Pergamon Press, New York.
- ICRP (International Committee on Radiological Protection) (1991) 1990 Recommendations of the International Commission on Radiological Protection. Pergamon Press.
- Imbus, C.E., Warner, J., Smith, E. *et al.* (1978). Peripheral neuropathy in lead-intoxicated sickle-cell patients. *Muscle Nerve* 1: 168-171.
- Inskip, M.J.; Atterbury, I. (1983) The legacy of lead-based paint: Potential hazards to "do-it-yourself" enthusiasts and children. In: Proceedings of an International Conference on Heavy Metals in the Environment: Heidleberg. CEP Consultants, Edinburgh: Vol. 1, 286-289.
-

-
- International Agency for Research on Cancer, IARC (1980) Lead and Lead Compounds. In: IARC Monographs on the Evaluation of the Carcinogenic Risk of Chemicals to Humans: Some Metals and Metallic Compounds 23: 325.
- International Agency for Research on Cancer, IARC (1982) IARC Monographs, Supplement 4: 149.
- International Agency for Research on Cancer, IARC (1987) IARC monographs on evaluation of carcinogenic risks to human: Overall evaluations of carcinogenicity: An updating of IARC monographs volume 1 to 42. Lyon., Franc 7: 230-232.
- Jelinek, C.F. (1982) Levels of lead in the United States food supply. *J. Assoc. Off. Anal. Chem.* 65: 942-946.
- Jenkins, G., Murray, C. and Hanna Thorpe, B. (1988) In: Lead in soil: Issues and guidelines. Environmental Geochemistry and Health Monograph Series 4, B. Davies and B. Wixson, eds. Northwood.
- Kang, H.K., Infante., P.F. and Carra, J.S. (1980) Occupational lead exposure and cancer. *Science* 207: 935-936.
- Kaspzrak, K.S., Hoover, K.L. and Poirier, L.A. (1985) Effects of dietary calcium acetate on lead subacetate carcinogenicity in kidney of male Sprague-Dawley rats. *Carcinogenesis* 6; 279-282.
- Kehoe, R.A. (1961) The metabolism of lead in man in health and disease: The normal metabolism of lead. (The Harben lectures, 1960). *J.R. Inst. Public Health Hyg.* 24: 81-97.
- Kimber, I., Jackson, J.A., Stonard, M.D. (1986) Failure of inorganic lead exposure to impair natural killer (NK) cell and T-lymphocyte function in rats. *Toxicol. Lett.* 31: 211-218.
- Kirkby, H. and Gynzelberg, F. (1985) Blood pressure and other cardiovascular risk factors of long-term exposure to lead. *Scand. J. Work. Environ. Health* 11:15-19.
- Kirkpatrick, D. and Coffin, D.E. (1974) The trace metal content of representative Canadian diets in 1970 and 1971. *Can Inst. Food Sci. Tech. J.* 56-58.
- Kirkpatrick, D.C. Conacher, H.B.S., Meranger, J.C. *et al.* (1980) The trace element content of Canadian baby foods and estimation of trace element intakes by infants.
- Koller, L.D. , Kerkvliet, N.I., and Exon, J.H. (1985) Neoplasia induced in male rats fed lead acetate, ethyl urea, and sodium nitrite. *Toxicol. Pathol.* 13:50-57.
-

-
- Koren, G., Nelson, C., Klien, J., Weiner, L. et al. (1990) Lead exposures among mothers and their newborns in Toronto. *Can. Med. Ass. J.* 142: 1241-1244.
- Lacey, R.F, Moore, M.R, and Richards, W.N. (1985) Lead in water, infant diet and blood: The Glasgow Duplicate Diet Study. *Sci. Total Environ.* 41, 235 -257.
- Lancranjan, I. (1975) Reproductive ability of workmen occupationally exposed to lead. *Arch. Environ. Health*, 30: 396.
- Landrigan, P.J., Gehlbach, S.H., Rosenblum, R.F., Shoults, J.M. et al (1975) Epidemic lead absorption near an ore smelter; The role of particulate lead. *N. Engl. J. Med.* 292:123-129.
- Langlois, P. (1992) The lead abatement project in South Riverdale. Draft Consultant's report. Occupational Health Centre, Queen's University.
- Lansdown, R.; Yule, W.; Urbanowicz, M.A.; Hunter, J. (1986) The relationship between blood lead concentrations, intelligence, attainment and behaviour in a school population: The second London study. *Int. Arch. Occup. Environ. Health* 57: 225-235.
- Lepow, M.L.; Bruckman, L., Rubino, R.A., Markowitz, S., Gillette, M.; Kapish, J. (1974) Role of airborne lead in increased body burden of lead in Hartford children. *Env. Health Perspect.* 7: 99-102.
- Levin R.(1987) Reducing Lead in Drinking Water: A Benefit Analysis. Washington, DC: United States Environmental Protection Agency (USEPA), 1986; USEPA report no. 230-09-86-019.
- Lilis, R., Fischbein, A., Diamond, S., Anderson, H.A., Selikoff, I.J., Blumberg, W. and Eisinger, J. (1977) Lead effects among secondary lead smelter workers with blood lead below 80 µg/100 mL. *Arch. Environ. Health*, 32: 256.
- Lin-Fu, J.S. (1972) Undue absorption of lead among children - A new look at an old problem. *N. Engl. J. Med.* 286: 702-710.
- Lin-Fu, J.S. (1992) Modern History of lead poisoning: A century of discovery and rediscovery in human lead exposure, Needleman, H. ed. CRC press. pp 23-43.
- Ludwig, G.D. (1977) Lead poisoning. Goldensohn, Eli S. and Stanley H. Appel (Ed.). Scientific approaches to clinical neurology, Vols. 1 and 2 XXII+992 (VOL. 1); X+1048p. (VOL. 2). Illus. Lea and Feviger: Philadelphia, Pa., USA.
-

-
- Lyngbye, T.; Hansen, O.N.; Trillingsgaard, A.; Beese, I.; Grandjean, P. (1990) Learning disabilities in children: significance of low-level lead exposure and confounding effects. *Acta Paediatr. Scand.* 79:352-60.
- Macpherson, A.S. (1987a) Children's Blood Lead Levels and environmental risk factors in the South Riverdale neighbourhood of Toronto and other areas of Ontario: A comparison study. City of Toronto, Department of Public Health, Toronto.
- Macpherson, A.S. (1987b) Memorandum from the City of Toronto Department of Public Health to the Board of Health, January 18, 1987. "Blood lead levels in Niagara Neighbourhood Children in 1984 and 1985 - A comparison to other Toronto children
- Maes, E.F., Swygert, D., Pascal, D., and Anderson, B. (1989) The contribution of lead in drinking water to levels of blood lead. I. A cross-sectional study. Unpublished report as cited in U.S. EPA 1991.
- Mahaffey, K.R., Rosen, J.F., Chesney, R.W., Peeler, J.T., Smith, C.M. and De Luca, H.F. (1982) Association between age, blood lead concentration, and serum 1,25-dihydroxycholecalciferol levels in children. *Am. J. Clin. Nutr.*, 35: 1327.
- Malcolm, D and Barnett, H.A.R. (1982) A mortality study of lead workers 1925 - 1976. *Br. J. In. Med* 39: 404-410.
- Mao, P. and Molnar, J.J. (1967) The fine chemistry of lead-induced renal tumours in rats. *Am. J. Pathol.* 50: 571-603.
- Marcus, A. (1985) Multicompartment kinetic models for lead, bone diffusion models for long-term retention. *Environ Res.* 36: 441-458.
- Marcus, W.L. (1986) Lead health effects in drinking water. *Toxicol. Ind. Health*, 2: 363.
- Marcus, A.H. (1989) Relationship between childhood blood lead and lead in water or diet. Battelle/Columbus Division, Arlington, MD. Report to USEPA/OTS AND ODW . Amended.
- Markovac, J., and Goldstein, G.W. (1988) Picomolar concentrations of lead stimulate brain protein kinase C. *Nature* 334: 71-73.
- MCSS (1992) Ontario Ministry of Community and Social Services. Memorandum communication
- McMichael, A.J., and Johnson, H.M. (1982) Long-term mortality profile of heavily exposed lead smelter workers. *J. Occup. Med.* 24; 375-378.
-

-
- McMichael, A.J.; Vimpani, G.V.; Robertson, E.F.; Baghurst, P.A.; Clark, P.D. (1986) The Port Pirie cohort study: Maternal blood lead and pregnancy outcome. *J. Epidemiol. Commun. Health* 40: 18-25.
- McMichael, A.J.; Baghurst, P.A.; Wigg, N.R.; Vimpani, G.V.; Robertson, E.F.; Roberts, R.J. (1988) Port Pirie cohort study: Environmental exposure to lead and children's abilities at the age of four years. *N. Engl. J. Med.* 319:468-475.
- Meranger, J.C. and Smith, D.C. (1972) The heavy metal content of a typical Canadian diet. *Can. J. Public Health* 63: 53-57.
- Meranger, J.C., Subramanian, K.S. and Chalifoux, C. (1979) A national survey for cadmium, chromium, copper, lead, zinc, calcium and magnesium in Canadian drinking water supplies. *Environ. Sci. Technol.*, 13: 707.
- Meranger, J.C., Subramanian, K.S. and Chalifoux, C. (1981) Survey for cadmium, basalt, chromium, copper, nickel, lead, zinc, calcium and magnesium in Canadian drinking water supplies. *J. Assoc. Off. Anal. Chem.*, 64: 44.
- Meranger, J.C., Subramanian, K.S., Langford, C.H. and Umbrasas, R. (1984) Use of an on-site integrated pump sampler for estimation of total daily intake of trace metals from tap water. *Intern. J. Environ. Anal. Chem.*, 17: 307.
- MOEE (1987) Ontario Ministry of the Environment. Review and Recommendations on a Lead in Soil Guideline. Report by the Lead in Soil Committee.
- MOEE (1988) Ontario Ministry of the Environment. Air Quality in Ontario: 1987, A Review of the Air Quality Monitoring Program, Air Resources Branch.
- MOEE (1990) Ontario Ministry of the Environment and Energy. Table of dry weight/fresh weight conversion factors. Unpublished. Phytotoxicity Assessment Section, Standards Development.
- MOEE (1991) Ontario Ministry of the Environment and Energy (MOEE). Air Quality in Ontario: 1989, A Review of the Air Quality Monitoring Program, Air Resources Branch.
- Moore, M.R. (1973) Plumbosolvency of waters. *Nature*, 243: 222.
- Moore, M.R., Meredith, P.A. and Goldberg, A. (1980) Lead and haem biosynthesis. In: Lead Toxicity. Edited by R.L. Singhal and J.A. Thomas. Urban and Schwarzenberg, Baltimore, p. 79.
-

-
- Moore, M.R., Goldberg, A., Fyfe, W.M. and Richards, W.N. (1981) Maternal lead levels after alterations to water supply. *The Lancet* (ii), 203.
- Moore, M.R.; Goldberg, A.; Pocock, S.J.; Meredith, A.; Stewart, I.M.; Macanespie, H.; Lees, R.; Low, A. (1982a) Some studies of maternal and infant lead exposure in Glasgow. *Scott. Med. J.* 27: 113-122.
- Moore, M.R.; Goldberg, A.; Bushnell, I.W.R.; Day, R.; Fyfe, W.M. (1982b) A prospective study of the neurological effects of lead in children. *Neurobehav. Toxicol. Teratol.* 4: 739-743.
- Morrow, P.E., Beiter, H., Amato, F., and Gibb, F.R. (1980) Pulmonary retention of lead: An experimental study in man. *Environ. Res.* 21: 373-384.
- Morse, D.L., Landrigan, P.J., Rosenblum, B.F. et al. (1979) El Paso revisited: Epidemiological follow-up of an environmental lead problem. *J. Am. Med. Assoc.* 242: 739-741.
- MTE (1988) Ministry of Treasury and Economics, Ontario Statistics
- Munoz, C., Garbe, K., Lilienthal, H., and Winneke, G. (1986) Persistence of attention deficit in rats after neonatal exposure to lead. *Neurotoxicology* (in press) as cited in Smith (1989).
- Mushak, P.; Davis, J.M.; Crochetti, A.F.; Grant, L.D. (1989) Prenatal and postnatal effects of low level lead exposure: Integrated summary of a report to the U.S. Congress on childhood lead poisoning. *Environ. Res.* 50:11-36.
- Nearing, J.N. (1987) Health Effects of inorganic lead with an emphasis on the occupational setting. Health Studies Service, Ontario Ministry of Labour.
- Needleman, H. (1992) Human lead exposure. H. Needleman, ed. CRC Press, London.
- Needleman, H.L.; Gunnoe, C.; Leviton, A.; Reed, R.; Peresie, H.; Maher, C.; Barrett, P. (1979) Deficits in psychologic and classroom performance of children with elevated dentine lead levels. *N. Engl. J. Med.* 300: 689-695.
- Needleman, H.L., Rabinowitz, M., Leviton, A. et al. (1984) The relationship between prenatal exposure to lead and congenital anomalies. *JAMA* 251: 2956-2959.
- Needleman, H.L., Geiger, S.K. and Frank, R. (1985) Lead and IQ scores: A reanalysis. *Science*, 227: 701.
-

-
- Needleman, H. and Bellinger, D.C. (1989) Type II fallacies in the study of childhood exposure to lead at low dose: A critical and quantitative review. In: Lead exposure and child development. An international assessment. M.A. Smith and A.I. Surs, eds. Kluwer Academic, Boston pp. 3-49.
- Needleman, H.L.; Gatsonis, C.A. (1990) Low-level lead exposure and the IQ of children. *JAMA* 263:673-8.
- Needleman, H.L., Schell, A., Bellinger, D., Leviton, A., Allred, E.N. (1990) The long-term effects of exposure to low doses of lead in childhood: An 11-year follow-up report. *N. Engl. J. Med.* 322:83-8.
- NFPA [National Food Processors Association] (1986) Can Manufacturers Institute, Inc. (1982) Comprehensive supplementary report covering further research, February 1980 through May 1983, on food borne lead in the diet of children. Available from: U.S. Department of Health and Human Services, Washington, D.C.; FDA docket no. 79N-0200.
- Ng, R. and Martin, D.J. (1977) Lead poisoning from lead-soldered kettle. *Can Med Assoc. J.* 116:508-510.
- Noqueiria, E. (1987) Rat renal carcinogenesis after chronic simultaneous exposure to lead acetate and N-nitrosodethyamine. *Virch. Arch. B* 53: 365-374.
- Nordstrom, S., Beckman, L. and Nordenson, I. (1979) Occupational and environmental risks in and around a smelter in northern Sweden: V. Spontaneous abortion among female employees and decreased birth weight in their offspring. *Hereditas* 90: 291-216.
- Nriagu, J. (1986) Lead contamination in the Canadian environment. Health effects of lead, Hotz, M.C.B. Royal Society of Canada. Commission on Lead in the Environment.
- Nutrition Canada (1977) Food Consumption Patterns report. Bureau of Nutritional Sciences, Health Protection Branch, Health and Welfare Canada.
- Nutrition Foundation (1982) Assessment of the safety of lead and lead salts in food. report of the Nutrition Foundation's Expert Advisory Committee pp.29-32.
- O'Flaherty, E.J., Hammond, P.B. and Lerner, S.I. (1982). Dependence of apparent blood lead half-life on the length of previous lead exposure in humans. *Fund. Appl. Toxicol.* 2: 49-54.
- O'Riordan, M.L. and Evans, H.J. (1974) Absence of significant chromosome damage in males occupationally exposed to lead. *Nature* 247: 50-53.
-

-
- Oberly, t.J., Piper, C.E., and McDonald, D.S. (1982) Mutagenicity of metal salts in the L5178Y mouse lymphoma assay. *J. Toxicol. Environ. Health* 9: 367-376.
- OMOH, Ontario Ministry of Health (1985) Blood lead and associated risk factors in Ontario children, 1984. Toronto, December 1985.
- Ong, C.N., Phoon, W., Lay, H. and Lim, H. (1985) Concentration of lead in maternal blood, cord blood, and breast milk. *Archives of Disease in Childhood* 60: 756-759.
- Otto, D., Benignus, V., Muller, K. *et al.* (1981) Effects of age and body lead burden on CNS function in young children. 1. Slow cortical potentials. *Electroencephalogr. Clin. Neurophysiol.* 52: 229-239.
- Otto, D.A., Benignus, V., Muller, K., Barton, C. *et al.* (1982) Effect of low to moderate lead exposure on slow cortical potentials in young children: Two year follow-up study. *Neurobehav. Toxicol. Teratol.* 4: 733-737.
- Oyasu, R., Battifora, H.A., Clasen, R.A., McDonald, J.H. and Hass, G.M. (1970) Induction of cerebral gliomas in rats with dietary lead subacetate and 2-acetylaminofluorene. *Cancer. Res.* 30: 1248-1261.
- Panova, A. (1972) Early changes in the ovarian function of women in occupational contact with inorganic lead. In: *Works of the United Research Institute of Hygiene and Industrial Safety. Bulgaria.* pp. 161-166.
- Piomelli, S., Seaman, C., Zullow, D., Curran, A. and Davidow, B. (1977) Metabolic evidence of lead toxicity in "normal" urban children. *Clin. Res.*, 25: 459A.
- Piomelli, S., Seaman, C., Zullow, D., Curran, A. and Davidow, B. (1982) Threshold for lead damage to heme synthesis in urban children. *Proc. Natl. Acad. Sci. USA.*, 79: 3335.
- Pirkle, J. L.; Schwartz, J.; Landis, J. R.; Harlan, W. R. (1985) The relationship between blood lead levels and blood pressure and its cardiovascular risk implications. *Am. J. Epidemiol.* 121: 246-258.
- Pocock, S.J., Shaper, A.G., Walker, M., *et al.* (1983) Effects of tap water lead, water hardness, alcohol and cigarettes on blood lead concentrations. *J. Epidemiol. Comm. Health.* 37, 1-7.
- Pocock, S.J., Shaper, A.G. *et al.* (1984) Blood lead concentration, blood pressure and renal function *Br. Med. J.* 289: 872-874.
-

-
- Pocock, S. J.; Shaper, A. G.; Ashby, D.; Delves, T. (1985) Blood lead and blood pressure in middle-aged men. In: Lekkas, T. D., ed. International conference: Heavy metals in the environment, v. 1; September; Athens, Greece. Edinburgh, United Kingdom: CEP Consultants, Ltd.; pp. 303-305.
- Pocock, S.J.; Ashby, D.; Smith, M.A. (1987) Lead exposure and children's intellectual performance. *Int. J. Epidemiol.* 16: 57-67.
- Pocock, S.J., Smith, M.A. (1989) Lead exposure and children's intellectual performance: the Institute of Child Health/Southhampton study. In: Lead Exposure and Child Development: An international Assessment. M.A. Smith, L.D. Grant and A.I. Sors, eds. Kluwer academic, Boston. pp. 149-165.
- Podrebarac, D.S. (1984) *J. Assoc. Off. Anal. Chem.* 68:1184-1197.
- Rabinowitz, M.B.; Wetherill, G.W.; Kopple, J.D. (1976) Kinetic analysis of lead metabolism in health humans. *J. Clin. Invest.* 58: 260-270.
- Rabinowitz, M.B.; Wetherill, G.W.; Kopple, J.D. (1977) Magnitude of lead intake from respiration by normal man. *J. Lab. Clin. Med.* 90: 238-248.
- Rabinowitz, M.B., Leviton, A., Needleman, H.L. (1986) Occurrence of elevated protoporphyrin levels in relation to lead burden in infants. *Environ. Res.* 39: 253.
- Rabinowitz, M. (1991) Toxicokinetics of bone lead. *Environ. Health Perspect.* 91: 33-37.
- Rai, K and Van Ryzin, J. (1985) A dose-response model for teratological experiments involving quantal responses. *Biometric* 41:1-9.
- Rasmussen, H. and Waisman, D.M. (1983) Modulation of cell function in the calcium messenger system. *Rev. Physiol. Biochem. Pharmacol.*, 95: 111.
- Regan, C. (1989) Lead-impaired neurodevelopment. Mechanisms and threshold values in the rodent. *Neurotox. and Teratotox.*, 11: 533-537.
- Rice, D.C. (1987) Primate research: Relevance to human learning and development. *Dev. Pharmacol. Ther.*, 10: 314.
- Rice, D. (1989) Behavioural effects of low-level developmental exposures to lead in the monkey. In: Lead exposure and child development: An international assessment. M.A. Smith, L.D. Grant and A.I. Sors, eds. Kluwer academic, Boston. pp. 427-439.
-

-
- Roberts, T.M.; Hutchinson, T.C.; Paciga, J.; Chattopadhyay, A.; Jervis, R.E.; Van Loon, J.; Parkinson, D.K. (1974) Lead contamination around secondary smelters: Estimation of dispersal and accumulation by humans. *Science* (Washington, D.C.) 196: 1120-1123.
- Robinson, T.R. (1974) 20-year mortality study of tetraethyl lead workers. *J. Occup. Med.* 16: 601-605.
- Robinson, G., Baumann, S., Kleinbaum, D. *et al.* (1985) Effects of low to moderate lead exposure on brain stem auditory evoked potentials in children. In: *Environmental Health 3: Extended abstracts from the 2nd international symposium on neurobehavioural methods in occupational and environmental health.*
- Roe, F., Boyland, E., Dukes, C.E., and Mithcley, B. (1965) Failure of testosterone or xanthopterin to influence the induction of renal neoplasms by lead in rats. *Br. J. Cancer* 19: 860-866.
- Roels, H.A., Buchet, J.P., Lauwerys, R., Hubermont, G., Bruaux, P., Claeys-Thoreau, F., Lafontaine, A. and Van Overschelde, J. (1976) Impact of air pollution by lead on the heme biosynthetic pathway in school-age children. *Arch. Environ. Health*, 31: 310.
- Roels, H.A.; Buchet, J-P.; Lauwerys, R.; Bruaux, P.; Claeys-Thoreau, F.; Lafontaine, A.; Verduyn, G. (1980) Exposure to lead by the oral and the pulmonary routes of children living in the vicinity of a primary lead smelter. *Environ. Res.* 22: 81-94.
- Rosen, J.F. (1985) Metabolic and cellular effects of lead: A guide to low level toxicity in children. In: *Dietary and environmental lead: Health effects* (K. Mahaffey, Ed.) Elsevier, New York, pp. 157-185.
- Rosen, J.F., Zarate-Salvada, C., Trinidad, E.E. (1974) Plasma lead levels in normal and lead-intoxicated children. *J. Pediatr.* 84: 45-48.
- Rosen, J.F., Chesney, R.W., Hamstra, A.J., De Luca, H.F. and Mahaffey, K.R. (1980) Reduction in 1,25-dihydroxyvitamin D in children with increased lead absorption. *N. Engl. J. Med.* 302: 1128.
- Rosen, J.F., Chesney, R.W., Hamstra, A.S. *et al.* (1980) Reduction in 1, 25-dihydroxyvitamin D in children with increased lead absorption. In: Brown, S., Orvis, D.S. eds. *Organ-directed toxicity. Chemical indices and mechanism.* New York, NY. Pergamon Press, 91-95.
- RSC (1986) Royal Society of Canada. Commission on Lead in the Environment (1986) *Lead in the Canadian Environment: Science and Regulation. Final Report.* Publ. by the Royal Society of Canada, September 1986.
-

-
- Rummo, J.H., Routh, D.K., Rummo, N.J. and Brown, J.F. (1979) Behavioural and neurological effects of symptomatic and asymptomatic lead exposures in children. *Arch. Environ. Health* 34: 120-124.
- Ryu, J.E., Ziegler, J.W. Nelson, S.E., and S.J. Fomon (1983) Dietary intake of lead and blood lead concentrations in early infancy. *Am. J. Dis. Child.* 137, 886-891.
- Schroeder, S.R.; Hawk, B.; Otto, D.A.; Mushak, P.; Hicks, R.E. (1985) Separating the effects of lead and social factors on IQ. *Environ Res.* 38:144-54.
- Schwartz, J. (1985a) Evidence for a blood lead-blood pressure relationship [memorandum to the Clean Air Science Advisory Committee]. Washington, DC: U.S. Environmental Protection Agency, Office of Policy Analysis. Available for inspection at: U. S. Environmental Protection Agency, Central Docket Section, Washington, DC; docket no. ECAO-CD-81-2 IIA.F.60.
- Schwartz, J. (1985b) Response to Richard Royall's questions on the blood lead-blood pressure relationships in NHANES II [memorandum to Dr. David Weil]. Washington, DC: U. S. Environmental Protection Agency, Office of Policy Analysis. Available for inspection at: U. S. Environmental Protection Agency, Central Docket Section, Washington, DC; docket no. ECAO-CD-81-2 IIA.C.5.
- Schwartz, J., Pitcher, H., Levin, R., Ostraw, B. and Nichols, A. (1985) Cost and benefits of reducing lead in gasoline: Final regulatory impact analysis, USEPA.
- Schwartz, J., Angle, C., Pitcher, H. (1986) Relationship between childhood blood lead levels and stature. *Pediatrics* 77:281-8.
- Schwartz, J. and Otto, D. (1987) Blood lead hearing thresholds and neurobehavioural development in children and youth. *Arch. Environ. Health* 42(3): 153-160.
- Science Advisory Board (1989) Review of lead carcinogenicity and EPA scientific policy on lead. EPA-SAB-EHC-90-001.
- Sedman (1987) The development of applied action levels for soil contact: A scenario for the exposure of humans to soil in a residential setting. State of California Department of Health Services, Toxic Substances Control Division, April 1987.
- Selevan, S.G., Landrigan, P.J., Stern, F., and Jones, J. (1985) Mortality of lead smelter workers. *Am. J. Epidemiol.* 122: 673-683.
- Seppalainen, A.M., Hernberg, S., Vesanto, R. and Kock, B. (1983) Early neurotoxicological effects of occupational lead exposure: A prospective study. *Neurotoxicology*, 4: 181.
-

-
- Sharp, D.S. (1987) Blood pressure and blood lead concentration in San Francisco MUNI bus drivers. PhD. Dissertation. University of California, Berkeley, CA. Ann Arbor, MI: University Microfilms International 1987: 93-95.
- Sharp, D.S., Benowitz, N.L., Osterloh, J.D., Becker, C.E., Smith, A.H., and Syme, S.L. (1990). Influence of race, tobacco use, and caffeine use on the relation between blood pressure and blood lead concentration. *American Journal of Epidemiology*. 131: 845-854.
- Sharp, D. S.; Becker, C. E.; Smith, A. H. (1987) Chronic low-level lead exposure: Its role in the pathogenesis of hypertension. *Med. Toxicol.* 2: 210-232.
- Sharp, D.S.; Osterloh, J.; Becker, C. E.; Bernard, B.; Smith, A. H.; Fisher, J. M.; Syme, S. L.; Holman, B. L.; Johnston, T. (1988) Blood pressure and blood lead concentration in bus drivers. In: Victory, W., ed. Symposium on lead-blood pressure relationships; April 1987; Chapel Hill, NC. *Environ. Health Perspect.* 78: 131-137.
- Sheppard, S. and Evenden, W. (1990) *J. Environ. Radioactivity* 11: 215.
- Sheppard, S. and Sheppard, M. (1991) Lead in boreal soils and food plants. *Water and Soil Poll.* 57-58:79-91.
- Sherlock, J.C., Ashby, D., Delves, H.T., Forbes, G.I., Moore, M.R., Patterson, W.J., Pocock, S.J., Quinn, M.J., Richards, W.N. and Wilson, T.S. (1984) Reduction in exposure to lead from drinking water and its effect on blood lead concentrations. *Human Toxicol.* 3: 383.
- Sherlock, J., Smart, G., Forbes, G.I. *et al.* (1982) Assessment of lead intakes and dose-response for a population in Ayr exposed to a plumbosolvent water supply. *Human Toxicol.* 1 , 115-122.
- Sherlock, J.C., and Quinn, M.J. (1986) Relationship between blood lead concentrations and dietary lead intake in infants: The Glasgow duplicate diet study. 1979-1980. *Food Additives and Contaminants*, 3:167.
- Siegal, M., Forsyth, B., Siegal, L. *et al.* (1989) The effects of lead on thyroid function in children. *Environ. Res.* 49: 190-196.
- Smith, M., Delves, T., Lansdown, R., Clayton, B. and Graham, P. (1983) The effects of lead exposure on urban children: The institute of child health/Southampton study. *Dev. Med. Child. Neurol.*, 25: suppl. 47.
- Smith, L. (1993) Blood lead levels in Ontario children. Public Health and Epidemiology Report Ontario (PHERO) Ontario Ministry of Health 4:7; 213-215 (July 30)
-

-
- Smith, M.A. (1989) The effects of low-level lead exposure on children. In: Lead Exposure and Child Development: An international assessment. M.A. Smith, L.D. Grant and A.I. Sors, eds. Kluwer academic, Boston. pp. 3-49.
- Statistics Canada (1987a) 1986 Census. Population and Dwelling Counts- Provinces and Territories Ontario
- Statistics Canada (1987b) 1986 Census. Profiles - Provinces and Territories, part 1.
- Stark, A.D.; Quah, R.F.; Meigs, J.W.; DeLousie, E.R. (1982) The relationship of environmental lead to blood-lead levels in children. *Environ. Res.* 27: 372-383.
- Staessen, J., Yeoman, W., Fletcher, A. *et al.* (1990) Blood lead concentrations, renal functions and blood pressure in civil servants. *Br. J. Ind. Med.* 47: 442-447.
- Steele, M.J., Beck, B.D., Murphy, B.L. and Strauss, H.s. (1990) Assessing the contribution from lead in mining wastes to blood lead. *Regulat. Toxicol. Pharmacol.* 11: 158- 190.
- Strehlow, C.D., Barltrop, D. (1987) Temporal trends in urban and rural blood lead concentrations. *Environ. Geochem. Health* 9(3-4): 74-79.
- Strenowsky, H. and Wellosowski, R. (1985) Lead and cadmium in breast-milk: Higher levels in urban versus rural mothers during the first 3 months of lactation. *Archives of Toxicology* 57: 41-45.
- Stoner, G.D., Shimkin, M.B. Troxell, M.C., and Terry, L.S. (1976) Test for carcinogenicity of metallic compounds by the pulmonary tumour response in Strain A mice. *Cancer. Res.* 36: 1744-1747.
- Stuik, E.J. (1974) Biological response of male and female volunteers to inorganic lead. *Int. Arch. Arbeitsmed.* 33,83-97.
- Sweeney, M.H., Beaumont, J.J., Waxweiler, R.J. and Halperin, W.E. (1986) An investigation of mortality from cancer and other causes of deaths among workers employed at an East Texas chemical plant. *Arch. Environ. Health.* 41: 23-28.
- Tanner, D.C. and Lipsky, M.M. (1984) Effect of lead acetate on N-(4'-fluoro-4-biphenyl)acetamide-induced renal carcinogenesis in the rat. *Carcinogenesis* 5: 1109-1113.
- Taylor, A. (1986) Metabolism and toxicology of lead. *Rev. Environ. Health* 6: 1-83.
- Tuppurainen, M., Wagar, G., Kurppa, K. (1988) Thyroid function as assessed by routine laboratory tests of workers with long-term lead exposure. *Scand. J. Environ. Health* 14: 175-180.
-

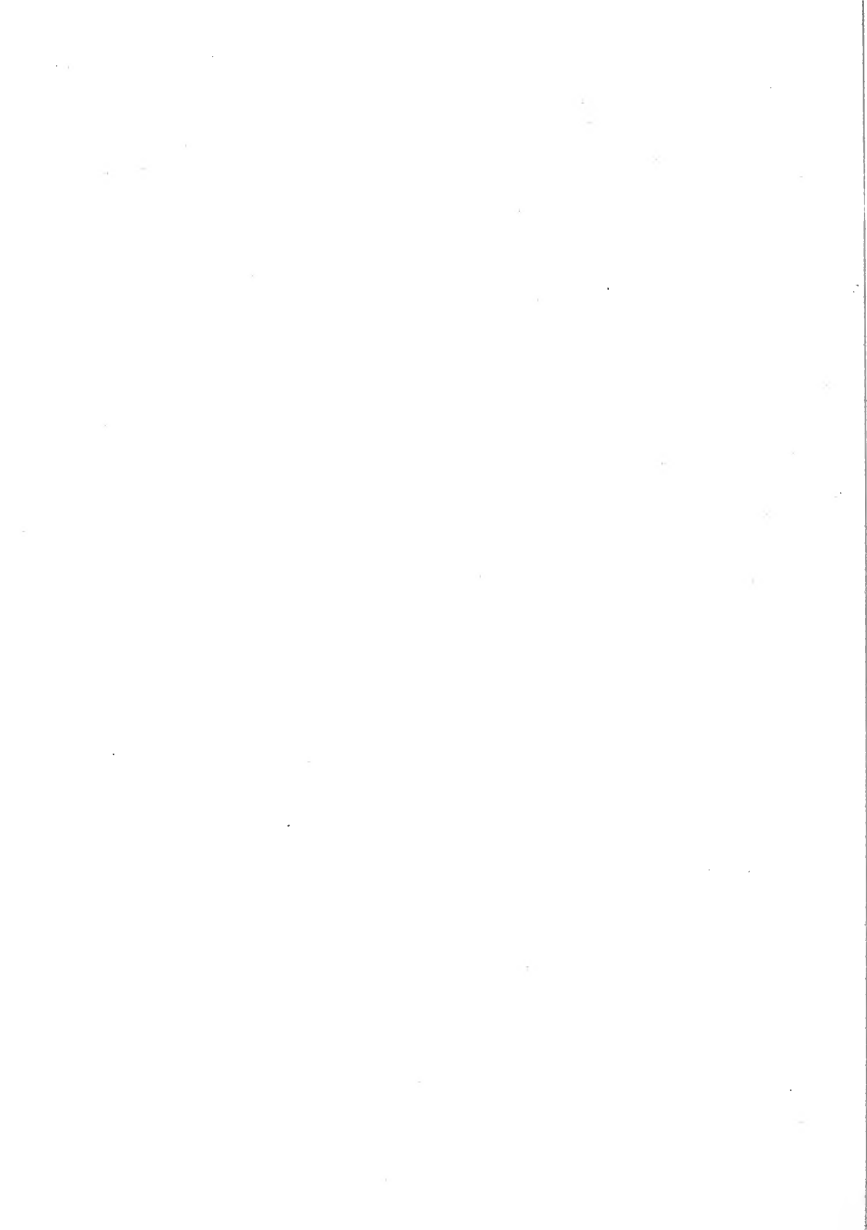
-
- United States Environmental Protection Agency, USEPA (1977) Air quality criteria for lead. Research Triangle Park, NC: Health Effects Research Laboratory, Criteria and Special Studies Office; U.S.EPA report no. EPA-600/8-77-017. Available from: NTIS, Springfield, VA; PB-280411.
- United States Environmental Protection Agency, USEPA (1980) Ambient water quality criteria for lead. USEPA 440/5-80-057. 151 pp. Avail. from NTIS, 5285 Port Royal Road, Springfield, Virginia 22161.
- United States Environmental Protection Agency (1984) Office of Policy and Analysis. Comments on issues raised in the analysis of the neuropsychological effects of low level lead exposure. Docket number ECAO-CDV-81-2.
- United States Environmental Protection Agency, USEPA (1986a) Environmental Criteria and Assessment Office. Air Quality Criteria for Lead. Washington, D.C. EPA-600/8-83.
- United States Environmental Protection Agency, USEPA (1986b) Lead effects on cardiovascular function, early development, and stature: an addendum to U. S. EPA Air Quality Criteria for Lead (1986). In: Air quality criteria for lead, v. 1. Research Triangle Park, NC: Office of Health and Environmental Assessment, Environmental Criteria and Assessment Office; pp. A1-A67; EPA report no. EPA-600/8-83/028aF. Available from NTIS, Springfield, VA; PB87-142378.
- United States Environmental Protection Agency (1988) Proposed Rule. Maximum Contaminant Level Goals and National Primary Drinking Water Regulations for Lead and Copper.
- United States Environmental Protection Agency, USEPA (1989a) Review of the National Ambient Air Quality Standards for Lead: Exposure Analysis Methodology and Validation. OAQPS Staff Report.
- United States Environmental Protection Agency, USEPA (1989b) Supplement to the 1986 EPA Air Quality Criteria Document for Lead.
- United States Environmental Protection Agency, USEPA (1989c) Evaluation of the Potential Carcinogenicity of Lead and Lead Compounds. Review. Office of Health and Environmental Assessment. Washington, D.C.
- United States Environmental Protection Agency, USEPA (1991) Technical Support Document for Lead. Internal Draft. Environmental Criteria and Assessment Office. Cincinnati.
- Van Esch, G. Van Gernderen, H., and Vink, H.H (1962) The induction of renal tumours by feeding of basic lead acetate to rats. *Br. J. Cancer* 16; 289-297.
-

-
- Van Esch, G.. and Kroes (1969) The induction of renal tumours by feeding basic lead acetate to rats. *Br. J. Cancer* 16: 289-297.
- Wallace, B. and Cooper, K. (1986) The citizen's guide to lead. NC Press Limited. Toronto.
- Walter, S., Yankel, A., von Lindern, I., (1980) Age-specific risk factors for lead absorption in children. *Arch. Env. Health* 35: 53-58.
- Weiss, S.T., Munoz, A., Stwin, A., Sparrow, D. and Speizer, F. (1986) The relationship of blood lead to blood pressure in a longitudinal study of working men. *Am. J. Epidemiol.* 123: 800-808.
- Wigg, N.R.; Vimpani, G.V.; McMichael, A.J.; Baghurst, P.A.; Robertson, E.F.; Roberts, R.J. (1988) Port Pirie cohort study: Childhood blood lead and neuropsychological development at age two years. *J. Epidemiol. Commun. Health* 42: 213-219.
- Wildt, K., Eliasson, R., and Berlin, M. (1983) Effects of occupational exposure to lead on sperm and semen. In: Clarkson, T.W., Nordberg, G.F., Salyer, P.R. eds. Reproductive and developmental toxicity of metals. Proceedings: Rochester, 1982 New York, NY: Plenum Press.
- Wilkins, J.R. and Sinks, T.H. (1984) Occupational exposures among fathers of children with Wilm's tumour. *J. Occup. Med.* 265:427-435.
- Wilson, T.W. and Card, R.T. (1986) Lead poisoning: unusual manifestation and unusual source. *Can. Med. Assoc. J.* 135: 773-775.
- Winder, C. (1989) Reproductive and chromosomal effects of occupational exposure to lead in the male. *Reprod. Toxicol.* 3(4): 221-234.
- Winneke, G., Hrdina, K.G. and Brockhaus, A. (1982) Neuropsychological studies in children with elevated tooth-lead concentrations. I. Pilot Study. *Int. Arch. Occup. Environ. Health*, 51: 169.
- Winneke, G., Kramer, U., Brockhaus, A., Ewers, U., Kujanek, G., Lechner, H. and Janke, W. (1983) Neuropsychological studies in children with elevated tooth-lead concentrations. II. Extended study. *Int. Arch. Occup. Environ. Health*, 51: 213.
- Winneke, G. and Kraemer, U. (1984) Neuropsychological effects of lead in children: Interactions with social background variables. *Neuropsychobiology*, 11: 195.
- Winneke, G.; Brockhaus, A.; Ewerts, U.; Kramer, U.; Neuf, M. (1990) Results from the European multicenter study on lead neurotoxicity in children: Implications for risk assessment. *Neurotoxicity and Teratology* 12:553-9.
-

-
- Wixson, B. (1989). Presentation on the Society of Environmental Geochemistry and Health (SEGH) Task Force approach for determining different soil lead clean-up levels. June 1, 1989.
- Wolf, A., Ernhart, C. and White, C. (1985) Intrauterine lead exposure and the status of the neonate. Heavy Metals in the Env., Int. Conf. Volume 1, 35-7, Lekkas, T., ed. CEP Consultants, Edinburgh.
- Wolff, M.S. (1983) Occupationally derived chemicals in breast milk. *Am. J. Ind. Med.* 4: 259-281.
- Wolnick, K., Fricke, F. et al. (1983) Elements in major raw agricultural crops in the United States: 1. Cadmium and Lead in lettuce, peanuts, potatoes, soybeans, sweet corn and wheat. *J. Agri. Food. Chem.* 31: 1240- 1244.
- World Health Organization, WHO (1977) Environmental Health Criteria 3. Lead.
- World Health Organization, WHO (1987) Joint FAO/WHO Expert Committee on Food Additives, 1986, 30th Meeting, Rome, 2-11 June, 1986, The Report.
- Yankel, A. J., Von, Lindern, I., Walter, S.D. (1977) The Silver Valley lead study: The relationship of childhood lead poisoning and environmental exposure. *J. Air Pollut. Control. Assoc.* 27: 763-767.
- Yule, W.; Lansdown, R.; Miller, I.; Urbanowicz, M. (1981) The relationship between blood lead concentrations, intelligence, and attainment in a school population: A pilot study. *Dev. Med. Child Neurol.* 23:567-76.
- Yule, W. and Rutter, M. (1986) Effects of lead on children's behaviour and cognitive performance: a critical review. In K. Mahaffey, ed. Dietary and Environmental Lead: Human Health Effects. Amsterdam, Elsevier.
- Zawirska, B. (1981) The role of the kidneys in disorders of porphyrin metabolism during carcinogenesis induced with lead acetate. *Environ. Res.* 24: 391-408.
- Zawirska, B. and Medras, K. (1968) Tumours and disturbances of porphyrin metabolism in rats resulting from chronic experimental lead intoxication. *Zentralblatt. Allgem. Pathol. Anatomie.* 111:1-12 as cited in USEPA (1989).
- Zawirska and Medras (1972) The role of the kidneys in disorders of porphyrin metabolism during carcinogenesis induced by lead acetate. *Arch. Immuno. Ther. Exp.* 20:257-272.
- Zelikoff, J.T., Li, J. H., Hartwig, A., et al (1988) Genetic toxicology of lead compounds. *Carcinogenesis* 9:1727-1732.
-

-
- Ziegler, E.E., Edwards, B.B., Jensen, R.L., Mahaffey, K.R., Fomon, S.J. (1978) Absorption and retention of lead by infants. *Pediatric Research* 12: 29-34.
- Zielhuis, R.L. (1975) Dose-response relationships for inorganic lead. *Int. Arch. Occup. Environ. Health*, 35: 1.
- Zimmermann-Tansella, C., Campara, P., D'Andrea, F., Savonitto, C. and Tansella, M. (1983) Psychological and physical complaints of subjects with low exposure to lead. *Human Toxicol.*, 2: 615.
- Zollinger, H. U. (1985) as cited in USEPA (1989b).

APPENDICES



APPENDIX A

ANALYTICAL METHODOLOGIES FOR LEAD

APPENDIX A ANALYTICAL METHODOLOGIES FOR LEAD

Several methods have been developed for the chemical analysis of lead in different environmental and biological media, including human tissues. The most commonly employed analytical techniques are atomic absorption spectrometry (AAS); X-ray fluorescence spectroscopy (XRF); anode stripping voltametry (ASV); and inductively coupled plasma-atomic emission spectroscopy (ICP-AES). AAS and ASV are considered the analytical methods of choice for lead (Grandjean and Olsen, 1984; U.S.EPA, 1985). The principal methodologies used to analyze lead in Ontario are described in this appendix.

A.1 SAMPLING AND ANALYSIS OF ENVIRONMENTAL MEDIA

The following sections briefly outline the sampling and analytical methods used in Ontario to determine the lead concentrations in various environmental media. At the Ontario Ministry of the Environment, air, water, soil and aquatic biota are routinely analyzed for lead and other heavy metals. The detection limits are summarized in Table A-1, together with the minimum measurable and minimum reliable values.

A.1.1 Air

Ambient air analysis is carried out using a standard high volume sampling technique with a glass fibre filter. This is followed by the determination of Total Suspended Particulate Matter and heavy metals analysis. The high volume sampler has a collection efficiency greater than 99%. Filters are prepared for AAS analysis by muffle furnace ashing, followed by nitric/hydrofluoric acid digestion. The extraction procedure is considered to give "total lead" (MOEE, 1979a,b).

A.1.2 Dust

Lead is emitted from point sources, such as secondary smelters, as a suspended particulate which eventually settles onto surfaces as dust. Monitoring of lead in dustfall is done by mounting a circular container (6 - 8 inches diameter, 16 -18 height) approximately 10 - 15 feet above the ground. Sample collection then occurs in a freefall nature and often times samples must be eliminated due to things other than dust being collected (eg. bird droppings). Sample contamination is a common occurrence when monitoring dustfall. 30 day monitoring periods are in place at which time the dust sample is sent to the ministry laboratory for analysis (weight, specific elements) (Wong, P., 1992).

Another type of dust occurs within households. Household dusts have been recognized as a potentially important source of lead exposure, particularly for children. Lead analysis of

household dust is not routinely done. At present, no standardized sampling methodology exists. The development of household dust sampling protocols is a new research area and there is considerable debate over acceptable methods.

TABLE A-1 LEAD ANALYTICAL DETECTION LIMITS

MATRIX	CONCENTRATION ¹		UNITS
Air	0.1 <W	0.5 <T	µg/m ³
Water			
Surface	0.005 <W	0.050 <T	µg/mL
Drinking	0.02 <W	0.2 <T	µg/mL
Sewage	0.02 <W	0.10 <T	µg/mL
Soil	2.0 <W	10.0 <T	µg/g
Vegetation	0.5 <W	2.5 <T	µg/g
Fish		0.60	µg/g
Blood		1.5	µg/L

¹W = minimum measurable value; T = minimum reliable value

Note: W is two-thirds of the standard deviation calculated on low level duplicate data. T is 5 times the W value. Where there are insufficient data to determine W, the instrument detection limit is reported.

A.1.3 Water

The analytical methodology used to determine lead in water depends on the source of the sample. Different protocols are employed in the collection and analysis of drinking water as opposed to surface waters.

Lead can leach into drinking water from lead piping present in the distribution systems or from solders used to connect copper piping. In households, three samples are routinely taken from the kitchen cold water tap to determine the maximum, minimum, and random concentrations. Samples are immediately preserved with nitric acid (MOEE, 1985). The samples are then analyzed directly by inductively-coupled plasma mass spectroscopy (ICP-MS) without further pretreatment (MOEE, 1988a).

Surface waters are collected in polyethylene terephthalate (PET) bottles and preserved with nitric acid. Prior to analysis by ICP, samples are reconcentrated by evaporation in the presence of nitric acid (MOEE, 1988b). The National Water Quality Laboratory of Environment Canada, however, uses reconcentration with hydrochloric acid prior to ICP analysis (CCIW, 1987a).

Sewage and sludge samples are analyzed by ICP atomic emission spectroscopy (MOEE, 1981a). A polarographic method may be used for the determination of lead at low concentrations in some aqueous matrices.

A.1.4 Soil

The protocol for collection of soil samples is contained in the MOEE Field Investigation Manual (MOEE, 1983). Different sample collection configurations may be applied to a given area: radii, quadrant and grid. In most cases, samples are taken at depths of 0-5 cm and 0-30 cm using a standard stainless steel core borer. Aliquots of dried sieved soil are subjected to a hot block aqua-regia digestion, then analyzed by ICP. This technique yields a recovery rate for lead of about 85% (MOEE, 1987a).

A.2 SAMPLING AND ANALYSIS OF ENVIRONMENTAL BIOTA

A.2.1 Vegetation

Vegetation samples are often collected in conjunction with soil samples, particularly in areas with point source lead emissions. Foliage samples are usually taken from the side of the tree or shrub facing the suspected contamination source. Both terrestrial and aquatic vegetation samples are analyzed by ICP following preparation by muffle furnace ashing and digestion with heated aqua-regia. Recovery of lead is about 94% (MOEE, 1987b).

A.2.2 Fish

Analysis of fish tissue samples is carried out by flame AAS after a perchloric-nitric acid hot block digestion (MOEE, 1987c). The Environment Canada protocol for lead in fish utilizes a hot block nitric acid - sulphuric acid- hydrogen peroxide ($\text{HNO}_3\text{:H}_2\text{SO}_4\text{:H}_2\text{O}_2$) digestion, followed by an ammonium pyrrolidine dithiocarbamate-methylisobutylketone extraction and flame AAS detection (CCIW, 1987b).

A.3 SAMPLING AND ANALYTICAL PROTOCOLS FOR BLOOD LEAD

A.3.1 Sampling Techniques

The protocol employed in the sampling and storage of blood samples is critical to the determination of blood lead concentrations.

Routine monitoring and epidemiological surveys of blood leads in Ontario typically use collection via a finger prick procedure (Duncan *et al.*, 1985; OMOH and MOEE, 1987;

MacPherson, 1985). The finger prick blood sample may be collected into a plastic microcontainer vial or a glass capillary tube. Because analytical techniques enable the detection of lead at very low levels, contamination of samples by even trace amounts of lead can increase the lead content of the sample considerably. Also, lead is ubiquitous in the environment, which greatly increases the potential for sample contamination. Considerable care must be exercised at each stage of sample collection and analysis.

Collection of a finger prick sample may be more liable to contamination than direct venepuncture, because of contamination of the skin surface at the puncture site. The generally higher analytical readings obtained using the finger prick method may also be attributed to the smaller sample size. The small size increases the relative impact of trace contamination. Direct transfer of blood into a microcontainer may introduce contamination through "scraping" of the puncture area with the microcontainer. Venepuncture is considered to provide the most reliable blood lead values, and is used in cases involving clinical referral. For routine monitoring and surveys, the finger prick method is used, mainly for efficiency. Precautions such as the thorough washing of children's hands and forearms, and the wearing of gloves by sampling personnel, are essential when taking capillary samples to minimize contamination.

The choice of anticoagulant may also affect analytical results (Grandjean and Olsen, 1984). Heparin at high concentrations can interfere with lead absorption in some AAS methods. EDTA may interfere with complex formation in other methods.

A.3.2 Quantitative Determination of Lead in Blood

The continuing need for blood lead monitoring has produced a number of analytical techniques which have been reviewed in detail (Grandjean and Olsen, 1984; U.S.EPA, 1986a). In general, the method will depend on the particular analytical lab performing the analysis and their technical capabilities. Based largely on considerations of efficiency, cost and reliability, atomic absorption spectrometry (AAS), in its various forms, is presently the most widely used method for routine determination of lead in whole blood. With regard to electrochemical techniques, anode stripping voltametry is the most sensitive and popular method. The most reliable and sensitive method for low levels of lead is isotopic dilution mass spectrometry (IDMS). However, IDMS is considered too costly and slow for general applications (Grandjean and Olsen, 1984).

In Ontario, blood sample analysis is carried out at the Ministry of Health and Ministry of Labour laboratories. Samples of 50 μ L are analyzed by graphite furnace AAS, utilizing Zeeman background correction (OMOH 1983a, OMOH, 1985b). Typically, a single blood sample from an individual is tested twice and the mean determined. In the 1985 Ontario Blood Lead Study, the mean difference between the two testings was not found to be statistically significant (Duncan *et al.*, 1985).

With regard to sample preparation, chelation/extraction methods have been found to be superior to acid digestion in some cases (U.S.EPA, 1986b). APDC was found to be a more effective chelating agent than either dimethylglyoxime or furildioxime. Although direct analysis of whole blood subsequent to haemolysis is considered simpler and less time-consuming than chelation extraction methods, direct analysis has increased potential for chemical interferences. As well, the analytical accuracy, precision and detection limit may be compromised relative to that achieved during ashing or chelation. The processed sample is then analyzed through AAS (flame and flameless) or ICP.

Quality control procedures are critical for accurate, reliable blood lead analyses. The analytical accuracy of the Ministry of Health Laboratory is verified on an on going basis through the Centers for Disease Control (CDC) lead analysis quality assurance program. The latter is the most comprehensive program of its type (Duncan *et al.*, 1985).

APPENDIX B

PHYSICAL AND CHEMICAL PROPERTIES OF LEAD

APPENDIX B PHYSICAL AND CHEMICAL PROPERTIES OF LEAD

Knowledge of the physical and chemical properties of lead is necessary to understand the element-media interaction and to develop the lead cycle. The relevant physical and chemical properties of lead are summarized in Table B-1 (Merck Index, 1983; U.S.EPA, 1988; Stokinger, 1981).

Lead (Pb) is a pale, silvery-grey metal, which is environmentally and biologically ubiquitous. Elemental lead exists as the following naturally occurring isotopes: ^{204}Pb (abundance, 1.4%), ^{206}Pb (25.2%), ^{207}Pb (21.7%), and ^{208}Pb (51.7%) (Merck Index, 1983). Isotopic ratios may vary considerably for different lead deposits (RSC, 1986). Twenty-four artificial radioactive isotopes of lead are known with atomic masses of 195-203, 205 and 209-214.

Lead has been widely used by humans for several thousand years. Metallic lead possesses a set of physical properties that have made it suitable for a variety of industrial applications. These properties include: high density; softness, malleability and high ductility; low melting point; resistance to corrosion; poor electrical conductivity; opacity to gamma and X-rays; radioactive absorptivity; and excellent sound attenuation characteristics.

Lead is rarely found in its elemental form. Like other metals, it will react to form compounds, complexes and alloys (Table B-2; Stokinger, 1981). It occurs most commonly as salts in mineral ores distributed throughout the earth's crust.

Lead is one of the Group IVA(14) elements, which also include germanium and tin. The metal possesses a face-centred cubic crystal structure.

TABLE B-1 PHYSICAL AND CHEMICAL PROPERTIES OF LEAD

CAS Registry No.:	7439-92-1
Synonyms:	Plumbum, Pigment metal
Atomic Number:	82
Atomic weight:	207.2
Melting point:	327.5°C
Boiling point:	1744°C (206 cal/g)
Vapour pressure:	1.77 mm Hg @ 1000°C
Specific gravity:	11.35 g/cm ³ @ 20°C
Odour:	None
Electronegativity:	1.8
Covalent radius:	1.44 Å
Electronic Structure:	[Xe]4f ¹⁴ 5d ¹⁰ s ² 6p ²

Due to the weakness of Pb-Pb bonding, lead has little tendency to catenate or form chains or clusters. It occurs in four valence states: Pb(O), Pb(I), Pb(II), and Pb(IV) of which Pb(II), the divalent state, is the most stable and predominant form. All four states are environmentally important, with the possible exception of Pb(I). Pb(II) readily forms strong covalent bonds with donor atoms such as sulphur and oxygen.

The chemistry of inorganic lead is well understood and has been thoroughly reviewed (Cotton and Wilkinson, 1988). The following observations on the general reactivity of lead are of environmental significance:

- it is sparingly soluble in hard, basic water and insoluble in saline solution;
- it reacts with acids to form ionic salts;
- it forms plumbites in alkali solution;
- it is attacked by halogens; and
- it is resistant to corrosion by dilute sulphuric, hydrofluoric and hydrochloric acids.

The solubility of lead in water varies with several factors such as temperature, pH and the presence of other compounds. Higher water temperatures increase lead solubility. The minimum solubility of lead is expected to occur at a pH of 9.8 and an alkalinity of approximately 40 mg/L as CaCO_3 . However, it has been found that the plumbosolvency of water in lead piping increases considerably outside the pH range of 6 - 8 (Moore, 1973). Other factors which may promote lead solubility in water are low organic content, low concentrations of suspended sediment and low concentrations of other metallic salts (Eisler, 1988).

The Pb(II) cation is partially hydrolysed in pure water to the hydroxide, Pb(OH)_2 . However, in natural water, this reaction is largely prevented by the presence of sulphates and carbonates, which form a protective coating on the lead surface.

With regard to the water solubility of its salts, lead chlorate, nitrate and acetate are water soluble; lead chloride is slightly soluble; and lead carbonate, chromate, phosphate, sulphate and sulphide are insoluble (U.S.EPA, 1988).

Although the divalent state, Pb(II), predominates, the tetravalent state, Pb(IV), is found in a number of halides, carboxylates, oxygen compounds and complexes. The ability of Pb(IV) to react with organic molecules gives rise to organolead compounds, such as tetramethyl and tetraethyl lead. The alkyl ligands in the latter organometallic complexes present singly or

monodentate binding sites to lead. However, other ligands, generally binding to Pb(II), can offer multiple binding sites, forming chelate rings (U.S.EPA, 1986). Chelation forms the basis for the clinical treatment of lead poisoning, using calcium disodium EDTA or D-penicillamine (Bondy, 1988). Lead is also capable of chelating with a number of biological molecules, including peptidyl groups and nucleotides.

The chemical bonding properties of lead may help to explain the biochemical basis for lead toxicity. The covalent/ionic character of lead bonds means that lead is a "soft" metal ion. It is therefore polarizable or deformable and tends to form stable covalent bonds with other "soft" atoms. It has been postulated that the binding of lead to "soft" atoms in biologically significant macromolecules, such as enzymes, may induce structural/conformational changes, leading to potentially toxic perturbations of function or activity (Turner *et al.*, 1985). The chemistry of organolead compounds is discussed in Appendix E.

TABLE B-2 SELECTED COMPOUNDS OF LEAD

TYPE	CHEMICAL FORMULA	CHEMICAL NAME	COMMON NAME(S)
Crystalline salts	$\text{Pb}(\text{CH}_3\text{COO})_2$	Lead acetate	Sugar of lead
	$2\text{PbCO}_3 \cdot \text{Pb}(\text{OH})_2$	Lead carbonate	Basic white lead
	PbS	Lead sulphide	Galena
	PbSO_4	Lead sulphate	Anglesite
	PbCrO_4	Lead chromate	Crocoisite, chrome yellow
	PbF_2	Lead fluoride	
	PbCl_2	Lead chloride	
	$\text{Pb}(\text{NO}_3)_2$	Lead nitrate	
	PbPO_4	Lead phosphate	
Oxides	PbO	Lead monoxide	Litharge
	PbO_2	Lead dioxide	
	Pb_3O_4	Triplumbic tetroxide	Red lead
	Pb_2O_3	Lead sesquioxide	
Tetravalent compounds			
Halogens	PbF_4	Lead tetrafluoride	
	PbCl_4	Lead tetrachloride	
Carboxylates	$\text{Pb}(\text{CH}_3\text{COO})_4$	Lead tetraacetate	
Organoleads	$\text{Pb}(\text{CH}_3)_4$	Tetramethyl lead (TML)	
	$\text{Pb}(\text{C}_2\text{H}_5)_4$	Tetraethyl lead (TEL)	

APPENDIX C
SOURCES AND EMISSIONS OF LEAD
INTO
THE ONTARIO ENVIRONMENT

APPENDIX C SOURCES AND EMISSIONS OF LEAD INTO THE ONTARIO ENVIRONMENT

Lead sources are both natural and anthropogenic in nature with the anthropogenic sources being much more predominant. In 1983 the median level of global lead emissions from natural sources was estimated to be 12×10^9 grams per year whereas anthropogenic sources emitted an estimated 332.35×10^9 grams per year.

The Canadian environment is contaminated by lead from almost entirely anthropogenic sources. In 1982, about 73,000 tonnes of lead were discharged into the Canadian environment. More than 83% was in the form of solid waste while atmospheric emissions accounted for nearly 16% and discharges into effluents less than 1%.

In 1982, the total lead contained in solid wastes in Canada was estimated to be 61,000 tonnes. This was six to seven times greater than the estimate for atmospheric emissions. Solid waste contributes the largest quantities, in large part from lead milling, smelting and refining industries. In 1982, milling of lead ores and primary smelting and refining contributed the most lead to solid wastes followed by secondary lead smelting, petroleum refining, manufacturing of pigments and explosives, sewage sludge, and leather tanning, respectively.

Atmospheric lead emissions have steadily decreased in the past decade. 1978, 1982 and 1987 inventories of lead reported lead emissions to the Canadian atmosphere to be 14534, 11466 and less than 6000 tonnes respectively. The projection for 1995 is 4000 tonnes.

The total Ontario lead emissions to the atmosphere were estimated to be 1504 tonnes in 1987, down from 3113 tonnes in 1982. The ten-year trend, 1980 to 1989, of ambient air lead levels in Ontario shows a significant decline (Figure D-1-1). This is largely due to the phasing out of leaded gasoline, which historically has been the major contributor of atmospheric lead. 1982 data show that lead emissions from gasoline accounted for 72% of lead emissions to the atmosphere whereas 1987 data shows leaded gasoline accounts for 43% of the total lead emitted in Ontario. Presently mining and milling processes are the greatest contributors to atmospheric lead with total lead emissions to the Ontario atmosphere estimated to be 550 tonnes/year. Other emitters of atmospheric lead are industrial processes including mining and milling of lead; copper, nickel, iron and steel production; ferrous foundries; storage battery production; and solid waste incineration.

The most significant local impacts of atmospheric lead emissions in Ontario are in the vicinity of the secondary lead smelters in Metropolitan Toronto. Together they constitute only about 0.5% of total lead emissions, but they are currently responsible for all of the measured exceedances of the 24-hour standard for particulate lead in air, and the 30 day point of impingement guidelines for lead in dust fall.

Effluents from municipal sewage treatment and industrial activities present a far smaller problem. The quantity of lead discharged in effluents represents only 5-6% of that emitted to the atmosphere. In 1982, Ontario discharged an estimated 125 tonnes of the total of 562 tonnes of lead in Canadian effluents. The largest source of lead in Ontario effluents, slightly more than two-thirds, was municipal sewage. In 1982, chemical manufacturing contributed 20% with the remaining 11% from smelting and refining, storage battery manufacture, primary iron and steel production, mining and milling, metal fabrication, foundries, refineries, and other miscellaneous industrial sources.

In Canada, the St. Lawrence River watershed receives the largest effluent discharge of lead, followed by the Great Lakes, Atlantic, Pacific, Shield, and Prairie watersheds. However, most of the lead in natural water bodies comes from atmospheric deposition rather than from industrial and municipal effluents.

Incinerators of municipal solid waste (MSW) emit about 25 times less lead than from all other known sources with most lead being found in ash. The lead ambient background level of incinerator emissions in air and soil are $0.7 \mu\text{g}/\text{m}^3$ and $16 \mu\text{g}/\text{g}$, respectively. Concentrations have been reported to range from $31 \text{ mg}/\text{kg}$ to $36000 \text{ mg}/\text{kg}$ likely due to the heterogeneity of the materials entering the incinerator.

A major source of lead in the Canadian environment is the lead-acid batteries used in motor vehicles. In 1986, there was 114,743 tonnes of lead in circulation on the roads (excluding batteries used by trucks, buses, trains, tractors, motorcycles, marine craft, and industry). In Canada, the battery industry accounts for approximately 50% of the total lead consumption. There are three large battery manufacturers in Canada sharing 65% of the market, of which 85% is in Southern Ontario.

Humans, however, may be at greater risk of exposure to lead from smaller scale sources of lead, quantitatively speaking. There are a number of consumer products that are potential sources for lead exposure. Sources warranting concern include lead-based paint, lead-soldered cans, and ceramics containing lead-based glaze. Minor exposure pathways include crystal, coffee cans, tobacco, wine, cosmetics, bread packaging, lead shot, pesticides, sewage sludge and fertilizer.

Lead-based paint has been recognized as a significant source of intermittent high-dose exposure. Since 1976 lead in paint is restricted to 0.5% (5000 ppm). Exposure could be significant in older homes, specifically those deteriorating or those being renovated, where lead-based paint may have once been used.

In 1985, lead-soldered seams were present in approximately one-third of the food cans used and in one-half of the beverage cans manufactured in Canada. As of 1988 a voluntary program has resulted in approximately 65% of lead-seamed cans being converted to welded cans. In some cases, such as beverage cans, conversion approached 100%.

Ceramics or other glazed containers used for storing, cooking, or serving food may be a source of lead. Lead may be leached from the ceramic due to inadequate glazing or from the glaze itself.

Drinking water systems have traditionally used lead and lead-containing products. Most small lead mains have been replaced. House plumbing can also be a source of lead where lead was previously used for all interior piping. Most of these old systems have been replaced. Unfortunately, the number of homes remaining that have interior lead piping is not known.

Elevated levels of lead in Ontario drinking water are primarily due to a combination of plumbosolvent water and lead plumbing or copper plumbing with lead solder. Levels of lead in source water do not contribute significantly. The Drinking Water Surveillance Program has reported flushed samples to be steadily declining from 3.0-4.0 µg/L (mean median) in 1985-1987 to 0.47 µg/L (mean average) in 1991.

APPENDIX C: SOURCES AND EMISSIONS OF LEAD IN THE ONTARIO ENVIRONMENT

C.1 INTRODUCTION

Lead is a naturally occurring metallic element, the fifth most commonly used metal in the world. In nature, the sources of lead are its ore deposits. Canada is the third largest producer of mined lead in the western world with 311,000 tonnes of primary lead being produced in 1984 (RSC, 1986).

Lead has many industrial uses. Hence, the mining, refining, processing, use, disposal, and recycling of lead and lead products all release some lead into the environment. Hence, both natural and man-made lead sources should be considered for exposure assessment.

In 1983 the median level of global lead emissions from *natural sources* was estimated to be 12×10^9 grams per year (Nriagu, 1989). Weathering and erosion of lead-containing surface ores are the largest source, followed by particles emitted during volcanic activity, forest fires and sea salt spray (Freedman and Hutchinson, 1981; Nriagu, 1986a, 1989). In Canada, lead is usually found in complex ores of lead and zinc sulphides (RSC, 1986). Estimates are not available for Canada specifically, but have been made for the global environment (Table C-1).

Lead emissions from *anthropogenic sources* greatly exceed emissions from natural sources. In 1983, atmospheric emissions from man-made sources were estimated to be 18 times greater than emissions from natural sources on a global scale (Nriagu, 1989). The median level of global lead emissions was estimated to be 332.35×10^9 grams per year. Estimates on a global scale are contained in Table C-1.

This chapter identifies and, where possible, quantifies, releases of lead into the environment. Ontario specific information is used where available. Alternatively, the best known information from other sources, primarily Canadian, is used. The first section of the appendix describes the nature of the lead emissions, while the remaining sections examine the most significant sources of lead exposure: industrial products, waste emissions, consumer and agricultural products, and drinking water.

TABLE C-1 GLOBAL EMISSIONS OF LEAD TO THE ATMOSPHERE IN 1983

SOURCE CATEGORY	RANGE (10 ⁶ kg yr ⁻¹)	MEDIAN (10 ⁶ kg yr ⁻¹)
<u>Natural Sources¹</u>		
Wind-borne soil particles	0.30 - 7.5	3.9
Sea salt spray	0.02 - 2.8	1.4
Volcanoes	0.54 - 6.0	3.3
Wild forest fires	0.06 - 3.8	1.9
Biogenic:		
Continental particulates	0.02 - 2.5	1.3
Continental volatiles	0.01 - 0.3	0.2
Marine	0.02 - 0.45	0.2
TOTAL, NATURAL EMISSIONS		12.0
<u>Anthropogenic Sources²</u>		
Coal combustion		
Electric utilities	0.78 - 4.65	
Industry and domestic	0.99 - 9.90	
Oil combustion		
Electric utilities	0.23 - 1.74	
Industry and domestic	0.72 - 2.15	
Pyrometallurgical non-ferrous metal production		
Mining	1.70 - 3.40	
Lead production	11.70 - 31.20	
Copper-Nickel production	11.05 - 22.10	
Zinc-Cadmium production	5.52 - 11.50	
Secondary non-ferrous metal production	0.09 - 1.44	
Steel and iron manufacturing	1.07 - 14.20	
Refuse incineration		
Municipal	1.40 - 2.80	
Sewage sludge	0.24 - 0.30	
Phosphate fertilizers	0.06 - 0.27	
Cement production	0.02 - 14.24	
Wood combustion	1.20 - 3.00	
Mobile sources		248.03
Miscellaneous	3.90 - 5.10	
Total, Anthropogenic Emissions	288.7 - 376.0	332.35
Total Emissions		344.00

¹ Source: Nriagu, 1989² Source: Nriagu and Pacyna, 1988

C.2 LEAD EMISSIONS INTO THE ONTARIO ENVIRONMENT

Anthropogenic sources account for nearly all of the lead released to the environment (Table C-1). In 1982, about 73,000 tonnes of lead were discharged into the Canadian environment. The relative contributions to different media are illustrated in Figure C-1. More than 83% was in the form of solid waste while atmospheric emissions accounted for nearly 16% and discharges into effluents less than 1% (Jaques, 1985a).

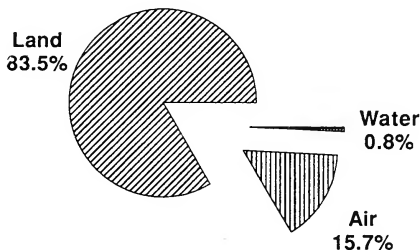


Figure C-1 Lead Release to Different Media
Canada - 1982

C.2.1 Solid Waste

Lead discharges in solid wastes have been studied far less than lead in atmospheric emissions and effluents. Although no Ontario-specific data are available, in 1982, the total lead contained in solid wastes in Canada was estimated to be greater than 61,000 tonnes (Table C-2) (Jaques, 1985a). This figure was six to seven times greater than the estimate for atmospheric emissions (Jaques, 1985a; Nriagu, 1986b).

For the Canadian environment, in 1982, milling of lead ores, and primary smelting and refining, contributed the most lead to solid wastes, followed by secondary lead smelting, petroleum refining, manufacturing of pigments and explosives, sewage sludge, and leather tanning, respectively (Jaques, 1985a). The lead content of municipal garbage varies from dump to dump according to the quantity of lead-soldered cans, used lead batteries, and other lead-containing items. The phase-down of leaded gasoline is decreasing the use of lead in petroleum refining, and its consequent release in the wastes from this industry.

TABLE C-2 ESTIMATED SOURCES OF LEAD IN SOLID WASTE: CANADA, 1982

SOURCE	AMOUNT (tonnes)
Tailings	38,000
Slags, drosses, and sludges (from primary smelting and refining of lead, zinc, copper, and nickel)	18,000
Sewage sludge	15
Spent lead shot	1,500
Secondary lead smelting	4,000
Petroleum refining	80
Explosives	30
Leather tanning	1
Inorganic pigments	100
Total	61,726

Source: Jaques, 1985a

C.2.2 Atmospheric Emissions

Historically, the primary source of atmospheric lead in Canada has been the combustion of leaded gasoline. In 1972, unleaded gasoline was introduced and the permissible concentration of lead in leaded gasoline was reduced to 0.77 g/L. The result has been a significant reduction in lead emitted to the air over the last 20 years and a sharp decrease in ambient air lead levels (MOEE, 1988c; MOEE, 1988d). 1978 and 1982 inventories of sources and releases of lead to the Canadian environment reported emissions to be 14534 tonnes and 11466 tonnes respectively (Jan and Sheffield, 1983; Jaques, 1985a). By 1987 lead emissions dropped to less than 6000 tonnes and the projection for 1995 is 4000 tonnes (Hilborn and Still, 1990).

In 1987, gasoline-powered motor vehicles were still the most significant source of lead emissions to the atmosphere, accounting for 43% of the total lead emitted in Ontario. This

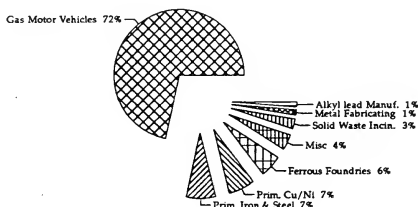


Figure C-2 Ontario Atmospheric Lead Emissions - 1982
Source: Jaques, 1985

represents a reduction from approximately 90% in the early 1970's and 72% in 1982. Automotive lead emissions originate from lead anti-knock compounds in gasoline (about 5% gaseous organolead compounds and 70% inorganic lead aerosol). Most lead deposition from automotive emissions occurs within 100 metres of the road, although resuspension of precipitated lead aerosol particles can extend the impacted area somewhat further (RSC, 1986). Other important sources of atmospheric lead are industrial processes including mining and milling of lead; copper, nickel, iron and steel production; ferrous foundries; storage battery production; and solid waste incineration (Jaques, 1985a; Jaques, 1985b).

The relative contributions of different industries to atmospheric lead in Ontario are illustrated in Figure C-2. This figure is based on 1982 data where lead emissions from gasoline accounted for 72%. Using the same data to be more indicative of present day, relative contributions were recalculated omitting gasoline emissions, assuming all other emission contributions have remained the same for the specified industries (Figure C-4). This indicates that mining and milling processes are the greatest contributors to atmospheric lead.

Recent data on approximate lead emissions to the atmosphere are available for Ontario (Table C-3) (MOEE, 1990a). These data indicated that industries where lead was measured specifically as lead and does not consider industries where the particulate matter emissions are not characterized for lead content. These data indicated an estimated total Ontario lead emissions to the atmosphere to be 323.9 tonnes in 1987 (MOEE, 1990a) down from 3113 tonnes in 1982 (Jaques, 1985a) (Figure C-4). This indicates a decline in atmospheric lead emissions to the Ontario environment, as confirmed by the downward ten-year trend in ambient air lead levels in Ontario (Appendix D1).

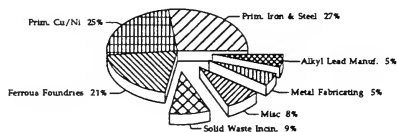


Figure C-3 Ontario Atmospheric Lead Emissions (Leaded Gasoline Omitted)
Adapted from: Jaques, 1985

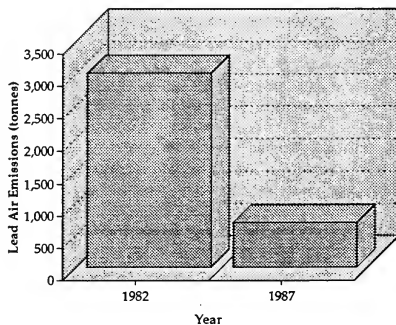


Figure C-4 Lead Air Emissions in Ontario
Source: Jaques, 1985, 1990

TABLE C-3 ATMOSPHERIC LEAD EMISSIONS IN ONTARIO

INDUSTRY	EMISSIONS (tonnes/year)
Non-Ferrous Smelters and Refiners	
Primary Smelters	211
Secondary Smelters	5.9
Automotive Industry	
Wet-cell Automotive Battery Production	51.1
Petrochemical Products Industry	
Metallic Additives	52.4
Waste Disposal Industry	
Semi-suspension Incinerator	2.40
Multichamber Incinerator	0.03
Fluidized-bed Incinerator	0.10
Multihearth Incinerator	0.90
Commercial & Industrial Incinerator	0.04
Total	323.87

Source: MOEE, 1990b

The most significant local impacts of atmospheric lead emissions in Ontario are in the vicinity of the three secondary lead smelters in Metropolitan Toronto: the Canada Metal Company, Toronto Refiners and Smelters Limited (now closed), and Tonolli Company of Canada Ltd. Together they constitute only about 0.5% of total lead emissions, but they are currently responsible for all of the measured exceedances of the 24 hour standard for particulate lead in air, and the 30 day point of impingement guidelines for lead in dust fall (MOEE, 1988c; MOEE, 1988d). It is likely that only the areas surrounding these point sources will have ambient air levels exceeding the guidelines.

C.2.3 Effluents

The quantity of lead discharged in effluents represents only 5-6% of that emitted to the environment (Jaques, 1985a; Nriagu, 1986b). In 1982, Ontario discharged an estimated 125 tonnes of the total of 562 tonnes of lead in Canadian effluents (Jaques, 1985a). The largest source of lead in Ontario effluents, slightly more than two-thirds, was municipal sewage (Table C-4). Sources of lead to the municipal system include industrial waste and run-off from roads. In 1982, chemical manufacturing contributed 20% with the remaining 11% from smelting and refining, storage battery manufacture, primary iron and steel production, mining and milling, metal fabrication, foundries, refineries, and other miscellaneous industrial sources (Jaques, 1985a; Jaques, 1985b).

In Canada, the St. Lawrence River watershed receives the largest effluent discharge of lead, followed by the Great Lakes, Atlantic, Pacific, Shield, and Prairie watersheds (Jaques, 1985a).

However, most of the lead in natural water bodies comes from atmospheric deposition rather than from industrial and municipal effluents (Evans and Rigler, 1985).

TABLE C-4 LEAD IN EFFLUENT DISCHARGES: ONTARIO, 1982

SOURCE	AMOUNT (tonnes)	% TOTAL
Municipal sewage	86	68
Chemical manufacturing	26	21
Smelting and refining	3	2
Storage battery manufacture	3	2
Primary iron and steel	3	2
Metal fabrication	1	1
Mining and milling	1	1
Foundries	<1	
Refineries	<1	
Miscellaneous	<1	
Total	125	100.0 %

Source: Jaques, 1985a

Currently in Ontario, the MOEE Municipal Industrial Strategy for Abatement (MISA) requires the monitoring of various mandatory and supplementary chemical parameters in the effluent discharge of identified effluent discharge sources. The program categorizes the effluent discharger into industrial or municipal sources. Presented in the subsequent tables is the long-term average loadings of lead from the individual industrial sectors and from the municipal sewage treatment plants. Because lead is not a mandatory chemical to be monitored not all companies listed under a particular sector will monitor for lead. Only the companies with lead monitoring information are presented below. With respect to the sewage treatment plants in the municipal category the values presented are the values where a value less than the detection limit is reported as the detection limit (also available are the data when the detection limit is taken to be zero).

TABLE C-5 MISA & INDUSTRIAL: IRON AND STEEL SECTOR AVERAGE
LOADING FOR PERIOD NOVEMBER 1, 1989 - OCTOBER 31, 1990
(ANNUAL AVERAGE)

COMPANY	LONG-TERM AVERAGE (kg/d)
ALGOMA STEEL	5
DOFASCO INC.	10.05
STELCO STEEL (Hilton Works)	5.36
STELCO STEEL (Lake Erie Works)	0.62
IVACO ROLLING MILLS	<0.01
LASCO	0.35
TOTAL SECTOR LOADINGS	21.38

Source: MOEE, 1991a

TABLE C-6 MISA INDUSTRIAL: ORGANIC CHEMICAL MANUFACTURING
SECTOR AVERAGE LOADING FOR PERIOD OF OCTOBER 1, 1989 -
MARCH 31, 1990 (ANNUAL AVERAGE)

COMPANY	LONG-TERM AVERAGE (kg/d)
Dow Chemical - Sarnia	28.5
Ethyl Canada - Corunna	20.0 (Inorganic) 0.2 (tetra-Alkyl Lead) 1.0 (Tri-Alkyl Lead)
Courtaulds Fibres - Cornwall	2.3
TOTAL SECTOR LOADINGS	50.8 (inorganic) 0.2 (tetra-alkyl lead) 1.0 (tri-alkyl lead)

Source: MOEE, 1992a

TABLE C-7 MISA INDUSTRIAL: INORGANIC CHEMICAL SECTOR AVERAGE
(ANNUAL AVERAGE)

COMPANY	LONG-TERM AVERAGE (kg/d)
General Chemical - Amherstburg	4.4
ICI - Cornwall	0.2
ICI Conpac - Cornwall	<0.1
Sulco Chemicals - Elmira	<0.1
TOTAL SECTOR LOADINGS	4.8

Source: MOEE, 1992b (unpublished data)

TABLE C-8 MISA INDUSTRIAL: METAL CASTING SECTOR AVERAGE
LOADING FOR PERIOD MAY 1, 1990 TO APRIL 30, 1991 (ANNUAL
AVERAGE)

COMPANY	LONG-TERM AVERAGE (kg/d)
Chrysler Canada - Etobicoke	<0.1
Canada Alloy Casting - Kitchener	<0.1
Canada Pipe Company - Hamilton	1.4
Kubota Metal, Fahramet Div. - Orillia	<0.1
Ford Motor Company - Windsor	4.4
Franklin Electric - Strathroy	<0.1
General Motors - St. Catharines	4.5
Haley Industries - Haley	<0.1
Richmond Die Casting	<0.1
Western Foundry Co. - Wingham	<0.1
TOTAL SECTOR LOADINGS	11

Source: MOEE, 1992c (unpublished data)

TABLE C-9 MISA INDUSTRIAL: PULP AND PAPER SECTOR AVERAGE LOADINGS (kg/d) FOR PERIOD JANUARY 1990 - DECEMBER 1990 (ANNUAL AVERAGE)

MILL	LONG-TERM AVERAGE (kg/d)
Abitibi-P (Ft. Will)	0.3
Abitibi-P (I. Falls)	1.4
Abitibi-P (Prov. P)	0.4
Abitibi-P (T. Bay)	0.5
Beaver Wood Fibre	0.1
Boise (Ft. Frances)	0.5
Boise (Kenora)	0.3
CP Forest (Dryden)	0.8
CP Forest (T. Bay)	1.2
Domtar (Cornwall)	3.9
Domtar (Red Rock)	3.0
Domtar (St. Cath)	0.1
Domtar (Trenton)	0.1
E.B. Eddy (Espanola)	2.4
E.B. Eddy (Ottawa)	0.1
James River - Marathon	2
Kimberly-C (Hunts.)	<0.1
Kimberly-C (St. Cath)	0.1
Kimberly-C (Terr. B.)	2.9
Macmillan Bloedel	0.9
Malette	0.6
Noranda Forest	0.3
Quebec and Ontario	0.6
Spruce Falls	2.6
St. Marys Paper	0.5
Strathcona	<0.1
Trent Valley (PBI)	<0.1
TOTAL SECTOR LOADINGS	25.9

Source: MOEE, 1991b, 1991c

TABLE C-10 MISA INDUSTRIAL: METAL MINING SECTOR AVERAGE LOADING FOR PERIOD OF FEBRUARY 1, 1990 - JANUARY 31, 1991 (ANNUAL AVERAGE)

COMPANY	LONG-TERM AVERAGE (kg/d) (kg/yr)
INCO, Refinery, Sudbury	0.1 45
Dickenson, Arthur W. White Mine	1.0 327
Teck - Corona, David Bell Mine	0.1 29.1
Hemlo Gold Mines, Golden Giant	0.12 21.7
Canamax, Maryhill Mine	<0.1 13.2
LAC Mineral S, Williams Mine	0.4 57
Rio Algom, Lacnor/Nordic	0.3 111
Rio Algom, Panel	0.4 138
Rio Algom, Quirke	1.1 378
Rio Algom, Stanleigh	1.1 351
TOTAL SECTOR LOADINGS	4.7 kg/d 1471 kg/yr

Source: MOEE, 1992d

TABLE C-11 MISA MUNICIPAL: SEWAGE TREATMENT PLANTS LOADINGS TO ONTARIO DRAINAGE BASINS

STP	LOADING (kg/d)
Brantford	1.6
Cornwall	1.5
Hamilton (Woodward)	9.6
London (Greenway)	2.8
London (Pottersburg)	0.4
Mississauga (Clarkson)	2.8
Mississauga (Lakeview)	11.1
Moore (Corunna)	0.1
Niagara Falls (Stamford)	1.4
Ottawa (Green Creek)	13.9
Pickering (Dufferin Creek)	5.4
Sarnia	5.8
Thunder Bay	3.7
Toronto (Highland Creek)	5.3
Toronto (Humber)	13.3
Toronto (Main)	48.6
Toronto (North)	1.0
Wallaceburg	0.2
Waterloo	1.4
Whitby (Pringle Creek)	0.1
Windsor (Little River)	1.1
TOTAL LOADINGS	131.1

Source: MOEE, 1992e (unpublished data)

C.3 SOURCES OF LEAD EXPOSURE IN ONTARIO

Quantification of lead emissions is not necessarily sufficient for a complete assessment of exposure. Certain sources of lead may, because of proximity or other factors, represent a greater risk of exposure. Such significant sources of lead have been identified and are discussed in detail in this section.

C.3.1 Lead in Emissions from Solid Waste Incinerators

Incinerators have been used in various capacities in Ontario for quite some time. The total amount of lead emitted from incinerators of municipal solid waste (MSW) is relatively small, about 25 times less than from all other known sources (Clement and Kagel, 1990).

Waste is categorized into two fractions: combustible and noncombustible. A U.S. study found, using a mass balance approach, that approximately 70% of waste is combustible. The average lead concentration in the input combustible fraction was 330 ppm (Law and Gordon, 1979).

Figure C-5 outlines the process of incineration in a simplified manner.

Incinerators have two types of emissions: solid ash residue and gaseous atmospheric emissions. The ratio of metal to organic emissions varies with the composition of the waste and the operating conditions, such as temperature or air distribution. Hence, from the point of view of control, a balance has to be maintained to keep hazardous emissions to a minimum. Some U.S. data on average lead emissions resulting from combustion of MSW, based on a mass balance approach, is contained in Table C-12. The total lead input from combustibles was 210 kg/week with a range of 70-970 kg/week (Law and Gordon, 1979).

TABLE C-12 LEAD EMISSIONS RESULTING FROM COMBUSTION OF MSW (US Data)

FINE BOTTOM ASH	FLY ASH	ATMOSPHERIC PARTICULATES
TOTAL ELEMENTAL OUTPUT (kg/wk)		
110 (60-160)	140 (80-200)	370 (270-470)

Source: Law and Gordon, 1979

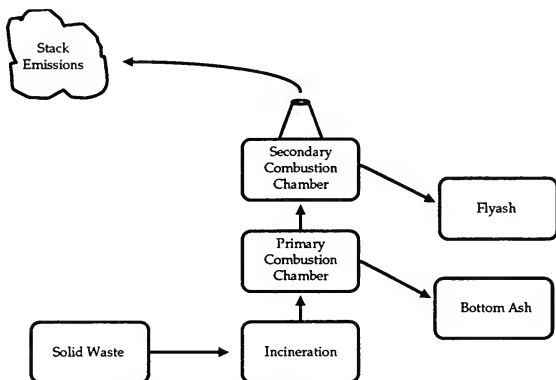


Figure C-5 Incineration Process (Gaseous and Solid State Emissions)

Lead may be found in both the ash and stack emissions from MSW incinerators. Because lead is relatively non-volatile, when incinerated, a relatively high proportion is found in ash and a relatively low proportion in gaseous emissions. In Ontario, the gas to ash partition coefficient for lead has been reported to be 1:300 (City of Toronto Board of Public Health, Report 2, 1988). The lead ambient background level of incinerator emissions in air and soil are $0.7 \mu\text{g}/\text{m}^3$ and $16 \mu\text{g}/\text{g}$, respectively (Medical Officer of Health, City of Toronto, 1985, as cited in CanTox, 1987).

C.3.1.1 Stack Emissions

Under normal operating conditions, the lead content in stack or gaseous emissions is low. In addition, incinerators are equipped with pollution control devices that minimize the release of lead into the atmosphere. The amount of lead emitted depends greatly on operating conditions. The National Incinerator Testing and Evaluation Program (NITEP) has tested performance in Canada under different conditions, providing an indication of expected emission rates (Tables C-13 and C-14).

The lead emission rates for three Ontario MSW incinerators and six biomedical waste incinerators are presented in Table C-15. All rates were within the Ontario Ministry of the Environment point of impingement (POI) value, set at $10 \mu\text{g}/\text{m}^3$ for lead emissions (MOEE Reg 308).

TABLE C-13 LEAD STACK EMISSIONS PER PERFORMANCE TEST MODE IN THE QUEBEC CITY INCINERATOR ($\mu\text{g}/\text{Sm}^3$)^{*}

GOOD CONDITIONS/ VARYING BURNING RATE			POOR CONDITIONS/ DESIGN BURNING RATE	
Low	Design	High	Low Temp	Poor Distribution
978	673	1599	2039	2495

Source: NITEP, June 1988.

TABLE C-14 LEAD STACK EMISSIONS PER PERFORMANCE TEST MODE IN THE PRINCE EDWARD ISLAND INCINERATOR ($\mu\text{g}/\text{Nm}^3$)^{*}

TEST MODE	NORMAL CONDITIONS	LONG CYCLE	HIGH 2° TEMP	LOW 2° TEMP
	14000	15000	15000	8400

Source: NITEP, Sept. 1989.

TABLE C-15 LEAD STACK EMISSIONS FROM ONTARIO MSW AND BIOMEDICAL WASTE INCINERATORS

LOCATION	AVERAGE STACK CONCENTRATIONS/ EMISSIONS	CALCULATED POI CONCENTRATIONS
MSW INCINERATORS		
Commissioners Street, Toronto ¹	1.261 tonnes/yr	N/A
Swaru, Hamilton ²	Actual 0.285 mg/m ³ Reference 0.534 mg/m ³	Impingement Concentration 0.050 $\mu\text{g}/\text{m}^3$
Ashbridges Bay, Toronto ¹	1710 kg/yr (adapted from 1986 data)	N/A

* $\mu\text{g}/\text{Sm}^3$ and $\mu\text{g}/\text{Nm}^3$ in the NITEP reports are used interchangeably. S (standards) and N (normal) denote operation at Standard Temperature and Pressure.

TABLE C-15 LEAD STACK EMISSIONS FROM ONTARIO MSW AND BIOMEDICAL WASTE INCINERATORS(Continued)

LOCATION	AVERAGE STACK CONCENTRATIONS/ EMISSIONS	CALCULATED POI CONCENTRATIONS
BIOMEDICAL INCINERATORS		
York Central Hospital, Toronto ³	2.2 mg/m ³	0.3 µg/m ³
Humber Memorial Hospital, Toronto ³	1.4 mg/m ³	0.2 µg/m ³
Womens College Hospital, Toronto ³	11.5 mg/m ³	3.1 µg/m ³
Toronto Western Hospital, Toronto ³	10.5 mg/m ³ 12.2 mg/m ³	0.2 µg/m ³ 0.2 µg/m ³
Oakville Trafalgar Hospital, Oakville ³	5.5 mg/m ³	0.1 µg/m ³
Victoria Hospital, London ⁴	Incinerator A Phase A 9.07 µg/m ³ B 116.3 µg/m ³ C 12.5 µg/m ³ Incinerator B Phase A 9.00 µg/m ³ B 40.3 µg/m ³ C 558.2 µg/m ³ Incinerator C Phase A 17.1 µg/m ³ B 78.2 µg/m ³ C 33.7 µg/m ³	N/A

Phase A - June 21, 1988 - June 24, 1988

Phase B - Nov 21, 1988 - Nov 25, 1988

Phase C - Feb. 21, 1989 - Feb 23, 1989

¹ City of Toronto Board of Health, Report 2, 1988.

² ORTECH International Report, October 1988.

³ Ozvacic, 1990.

⁴ National Incinerator Testing and Evaluation Program, September 1989.

Lead is found in ash residue in both the organic and inorganic form. Ash residue may be classified into bottom ash and fly ash or precipitator ash. *Bottom ash* is the portion of waste that comes from the primary chamber. It is normally transported to a monofill or it may be reused as road base material or light weight aggregate (Rod Fogget, personal communication; Korzun and Heck, 1990). *Fly ash* is the particulate removed from combustion flue gases bound for the smoke stack but captured by pollution control equipment (Reg 309). Fly ash generally contains higher levels of both organic and inorganic lead. Because of this difference in concentrations (Table C-16), Regulation 309 under the Environmental Protection Act requires that fly ash be kept separate from bottom ash and be disposed of in separate approved locations.

The lead content varies greatly with different types of ash. Concentrations have been reported to range from 31 mg/kg to 36000 mg/kg (U.S.EPA, as cited in Korzun and Heck, 1990). This variance is due to the degree of heterogeneity of the materials entering the incinerator. In order to determine whether the ash is hazardous, a leachate extraction test is done, as outlined in Reg 309. The reliability of the test is not consistent (Clement and Kagel, 1990). The lead concentrations in leachate from some Canadian incinerators are reported in Table C-17.

TABLE C-16 CONCENTRATIONS OF LEAD IN ASH RESIDUE OF SOME CANADIAN INCINERATORS

	BOTTOM ASH	FLY ASH	REFERENCE
Ashbridges Bay, Toronto	Total Ash	1.2 g/kg	Metro Works Dept., 1987 as cited in the City of Toronto Board of Health, Report 2, 1988
Quebec City	1760 ppm	Boiler 7480 ppm Precipitator 21100 ppm	NITEP, June 1988
Victoria Hospital, London ¹	1970 ppm	Boiler 23800 Economizer 24000	NITEP, September 1989
PEI	2600 ppm	Boiler 8500 ppm Economizer 8000 ppm	NITEP, September 1985
Swaru, Hamilton	1320 ppm	3200 ppm	NITEP, July 1989

TABLE C-17 CONCENTRATIONS OF LEAD IN LEACHATE AT SOME CANADIAN INCINERATORS (ppm)

	BOTTOM ASH	FLY ASH	REFERENCES
Ashbridges Bay, Toronto	Total Ash<0.05		Metro Works Dept., 1987 as cited in the City of Toronto Board of Health, Report 2, 1988
Victoria Hospital, London ¹	6.83*	Boiler 4.15 Economizer 19.3*	NITEP, September 1989
Quebec City	8.03*	Boiler 39.3* Precipitator 35.9*	NITEP, June 1988
Swaru, Hamilton	<0.05	0.67	NITEP, July 1989

*Exceeds the Ontario limit (5.0 ppm.)

Three different methods are available to treat fly ash to reduce the concentration of lead in the leachate prior to disposal at the monofill. All are fairly successful as evidenced by the results from the Quebec City incinerator (Table C-18).

TABLE C-18 REDUCTION OF LEAD LEVELS IN TREATED FLY ASH (QUEBEC CITY INCINERATOR).

	ESP ASH	ESP/CKD	ESP/BFS	ESP/FA
Leachate (ppm)	11.2	<0.05	<0.05	<0.05
Emissions (ppm)	8600	5450	5650	5350

ESP = electrostatic precipitator FA = fly ash

CKD = cement kiln dust BFS = blast furnace slag

Source: NITEP, July 1989.

Another suggested alternative would be to sort waste prior to incineration in order to eliminate any material that might increase the lead content of the ash residue. However, it is difficult to determine what material produces hazardous waste upon incineration. Removal of noncombustible products prior to incineration does reduce the heavy metal content of incinerator ash. Ninety-eight per cent of lead in MSW is contained in noncombustible products, the major contributor being lead-acid batteries. US data from 1986 showed lead-acid batteries to be 65% of lead in MSW. After removal of the non-combustible portion, most of the remaining lead is in plastics and other pigments (Franklin Associates, 1989).

A major source of lead in the Canadian environment is the lead-acid batteries used in motor vehicles. A typical passenger car battery weighs 20 kg and is approximately 50% lead (Sypher Mueller Report, 1988). In 1986, there were over 11,000,000 cars registered in Canada resulted in over 100,000 tonnes of lead in circulation on the roads. If the batteries used by trucks, buses, trains, tractors, motorcycles, marine craft and industry had been included, this estimate would have almost doubled (Statistics Canada as cited in the Sypher:Mueller Report, 1988). The estimated scrappage rate of batteries is four million units per year, equivalent to 75 000 tons of lead.

Canada's largest user of lead is the battery industry which accounts for approximately 50% of the total lead consumption (RSC, 1986). There are three large battery manufacturers in Canada: Varta (Ontario, Quebec and Manitoba); GM Delco (Ontario); and Batttronics (Ontario and Quebec). They share 65% of the market, of which 85% is in Southern Ontario. The import of new batteries for replacement almost equals the export. Canada imports all replacement batteries for motorcycles, snowmobiles and all-terrain vehicles (Sypher Mueller Report, 1988).

In 1986, Canadian smelters imported 61,530 tonnes of lead scrap (Statistics Canada as cited in the Sypher:Mueller Report, 1988). Scrap batteries are imported in addition to replacement batteries because of an insufficient supply of used batteries for Canadian smelters. Also, transportation costs are low. Import rates from the United States can be as high as 50% per year (Sypher Mueller Report, 1988). In 1987, Tonolli received 40% of its scrap batteries from Ontario, 10% from other Canadian provinces and nearly 50% from the United States (Coschi, personal communication).

A battery typically consists of 50% lead from a primary smelter operation and 50% secondary recycled lead from a secondary smelter operation where lead is reclaimed from used lead-acid batteries. Although the life of a battery is estimated to be four years, its lead can be recycled and subsequently used in other batteries or in other industries. The life-cycle of a lead-acid battery is illustrated in Figure C-6.

A typical battery recycling operation is shown in Figure C-7. Battery recycling consists of the physical breakage of the batteries; wet screening for paste separation; paste desulphurization; and metallic fraction separation from ebonite, polyvinyl chloride and polypropylene. The final products include: grids and poles; desulphurized paste; anhydrous sodium sulphate; and polypropylene (Tonolli, 1986). Polypropylene may be sold to plastic recyclers and sodium sulphate to detergent manufacturers, while the desulphurized paste is discarded. The grids and poles are recycled to refined lead, alloy lead and solder.

The secondary lead smelter is one of the most efficient recycling industries. It claims to have a 98% recovery efficiency in the recycling of its raw materials (RSC, 1986). In 1987, the Canadian lead industry recycled 11,330,000 batteries for an output of 112,000 tonnes of lead (Sypher Mueller, 1988).

Almost all of the spent lead is potentially recyclable. The U.S. recycling rates of lead-acid batteries are given in Figure C-8. In the peak year of 1979, nearly 90% of old batteries were recycled, but in 1984 and 1985, the rates decreased to 66% and 58%, respectively (Coschi, personal communication). This decline may be attributed to a lack of incentive for consumers who do not receive any compensation for used batteries and may even be charged a fee. As a result, many lead-acid batteries end up in the solid waste stream. Consumers may dispose of batteries in municipal landfills, backyards or dumps where lead corrosive acids may be present. Lead is resistant to sulphuric, sulphurous, phosphoric and chromic acids, but it is moderately soluble in hydrochloric and hydrofluoric acids. It is highly soluble in nitric, acetic, formic and citric acids (Jaques, 1985a). This may result in leaching of lead from the soil into ground water.

New regulations lowering the limits for lead in gasoline and paint, together with the low incentive to recycle, will make lead-acid batteries the major source of lead in the environment, with most of it being contained in municipal solid waste. In 1986, 65% of the lead in U.S. municipal solid waste was from lead-acid batteries (Franklin Associates, 1989).

A waste battery is defined under Ontario Regulation 309 as a discarded, disused, broken, spent or worn out lead-acid battery. As of 1985, dealers of used batteries, scrap yards and battery breakers were required to obtain certificates of approval, registration and manifesting (MOEE, 1990c). Unfortunately, the new regulation has discouraged battery collectors resulting in a decline in battery recycling in Ontario (Killackey, personal communication). A new amendment implemented in 1988 exempts waste battery collectors.

Secondary lead smelters are regulated under federal law (Table C-19). They must also satisfy the ambient pollution and ambient air quality criteria contained in Ontario regulations (Table C-20).

Waste water emissions from Ontario smelters may be discharged to municipal sewers, and are regulated by the appropriate municipality's sewer-use by-law (Coschi, personal communication). Lead levels in effluent from the Tonolli plant are 4-5 ppm with a projected decrease to 1 ppm upon installation of a new process to reduce waste by reclaiming all components of scrap batteries (Tonolli, 1986).

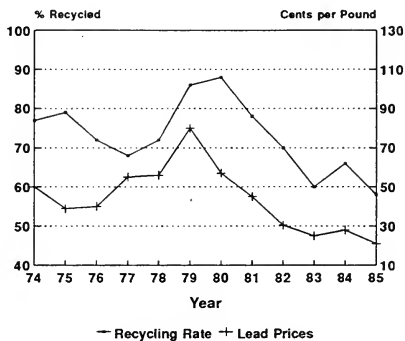


Figure C-8 U.S. Battery Rates vs. Lead Prices
Source: Tonolli, 1986

TABLE C-19 FEDERAL AIR EMISSION REGULATION

PARAMETER	OPERATIONS	MAXIMUM CONCENTRATION
Particulate matter	Blast furnaces Cupolas Reverberatory furnaces	46 mg/m ³
	Holding furnaces Kettle furnaces Lead oxide mills Scrap handling Crushing Furnace tapping Furnace slagging Furnace cleaning Casting	23 mg/m ³

Source: Canada Gazette.

TABLE C-20 ONTARIO AMBIENT AIR CRITERIA

AIR	CONCENTRATION
24 hour ambient criteria	5.0 $\mu\text{g}/\text{m}^3$
30 day geometric mean	2.0 $\mu\text{g}/\text{m}^3$
Point of impingement 1/2 hour average	10.0 $\mu\text{g}/\text{m}^3$

An increase in production costs and a decrease in the price of lead has led to the closure of many primary and secondary smelters in Canada and the United States. Between 1983 and 1985, the number of American smelters decreased from 80 to 23 (Coschi, personal communication). The existing operations are predominantly located in or near large cities where scrap can be obtained in sufficient quantities for continuous operation (Sypher Mueller Report, 1988). The capacity of the six Canadian secondary smelters currently in operation are given in Table C-21.

TABLE C-21 SECONDARY LEAD SMELTER CAPACITY

SMELTER	CAPACITY (tons/year)
Tonolli Canada Ltd, Ontario	120,000
Canada Metals Ltd, Ontario	10000-12000
Canada Metals Ltd, Manitoba	3500-4000
Nova Pb Inc., Québec	50,000
Metalex Products LTD., British Colombia	5,000
Cominco, British Colombia	8,000

Source: Sypher:Mueller Report, 1988; Tonolli, 1986

C.3.3 Consumer Products

A number of consumer products contain lead. Hence, these may be a source of lead emissions into the environment or a source of lead exposure for humans. Regulations limiting the lead content of many products are now in effect. The public is considerably more aware of the potential hazards of lead-containing products than in the past. However, items obtained second-hand or purchased prior to the regulations could present a risk of exposure to lead.

In 1985, lead-soldered side seams were present in approximately one-third of the food cans used and in one-half of the beverage cans manufactured in Canada (RSC, 1986). As of 1988, a voluntary program to eliminate lead-soldered cans has resulted in approximately 65% of Canadian manufacturers converting to welded cans (Huston, 1990). In some cases, such as beverage cans, conversion approaches 100%. The conversion is expensive and many small canners, such as fish canners who are in operation only a few weeks each year, have not changed over. More than half of imported food cans have lead-soldered seams (J. Nixon, personal communication).

The Canadian Food and Drug Act sets limits on the lead content of certain canned food products to be sold in Canada. The legislation applies to both domestic and imported goods (Table C-22). In addition, Health and Welfare Canada has worked with the canning industry to improve soldering techniques. This has led to improved application of solder and has reduced lead leaching into canned food.

Health and Welfare Canada conducts two types of food products surveys: one to determine compliance with the Food and Drug Act and the second, to gather data on 20 common food classes. If high levels of lead are found in a product not included under the Act, a review is done to determine if the product is safe and whether it can be sold. Of the 771 samples tested for lead, four exceeded the 10 ppm lead limit (J. Nixon, personal communication).

One alternative under consideration by Health and Welfare Canada would be to set a limit of 0.5 ppm lead for all tinned goods not specifically listed in the Food and Drug Act (J. Nixon, personal communication).

TABLE C-22 CANADIAN LIMITS ON LEAD CONTENT IN CANNED FOOD PRODUCTS

TYPE OF CANNED FOOD	LIMIT ON LEAD CONTENT ($\mu\text{g/g}$)
Concentrated Infant formula evaporated and condensed milk	0.15
Ready-to-serve infant formula	0.08
Fruit juices	0.20
Tomato paste and sauce	1.50
Ready-to-serve beverages in sealed containers other than mineral water	0.20

Source: RSC, 1986.

Lead-based paint has been recognized as a major source of high-dose exposure and as a cause of symptomatic lead poisoning in children in certain parts of the U.S.A. Lead is used in paints as a pigment, a dispersing agent, or a drying agent. It also stabilizes tannins, thus preventing discolouration of wood surfaces. In the past, leaded paints in the U.S.A. may have contained up to 500,000 ppm lead. However, since 1977, household paints, by regulation, must not contain more than 600 ppm lead (CDC, 1985).

In Canada, the Hazardous Products Act prohibits the use of lead pigments in interior consumer paints and in the paint applied to children's toys and furniture. Lead, however, may be added to such paints for other purposes; for example, to facilitate dispersal or to speed drying. The Act restricts the amount of lead added for such purposes to less than 0.5% (5000 ppm) metal relative to the total solids in the coating (RSC, 1986). Paint used on other surfaces may contain up to 5000 ppm lead (RSC, 1986). Most paints used in Canada contain less than 600 ppm lead, but a minority may have a significantly higher lead content (J. Gibson, personal communication).

The two lead pigments used in Canada are lead oxide and lead chromate. The former is used primarily for corrosion protection of steel structures, while the latter is used in vehicle paints, road traffic markings, printing inks, and plastics (Gray, 1985, cited in RSC, 1986). Erosion of lead-painted structures, vehicles and road surfaces releases lead particles into the environment. Sandblasting of lead-painted outdoor structures, such as bridges, can greatly increase lead concentrations in local terrestrial and aquatic environments.

Paint chips from peeling exterior or interior house paint containing lead also release lead into the environment, and have been associated with numerous cases of lead poisoning in U.S. children. People appear to be at greatest risk of lead exposure when lead-painted surfaces are sanded. Large quantities of airborne lead particles may be produced. One study found indoor air concentrations of 550 $\mu\text{g Pb}/\text{m}^3$ after 5 minutes of sanding a windowsill coated in paint with a lead content of 0.8 - 0.9 mg/cm^2 (Feldman, 1978, cited in Elias, 1985). Consumers may also be at risk during the application of lead-based paint; spray painting can generate high concentrations of lead-containing mist.

Present Canadian regulations are currently being reviewed with an aim to lower the legal concentration of lead in paint. A lead level of 0.06% is being considered, as well as removal of the distinction between interior and exterior paints (J. Gibson, personal communication). Another alternative may be to introduce a use-specific classification system; for example, a higher lead concentration might be allowed for paints used on bridges or roads.

Ceramics or other glazed containers used for storing, cooking, or serving food may be a source of lead. Lead may be leached from the ceramic due to inadequate glazing or from the glaze itself. A fatal case of lead poisoning from a ceramic occurred in Canada about 20 years ago: a child died after drinking apple juice stored in a vase (J. Gibson, personal communication). In the U.S.A., two recent cases of acute lead poisoning were reported as the result of consuming beverages from glazed cups and glasses (Lecos, 1987).

The U.S. Food and Drug Administration (1980) and the International Organization for Standardization (ISO) have set a three-tiered standard for ceramics, based on the depth or size of a ceramic good (Elias, 1985; U.S.EPA, 1986). The U.S. FDA is now proposing a reduction in the allowable limit to 0.1 ppm lead in the acid leachate test for ceramics. In the acid leachate test, ceramics and glazed containers are filled with 4% acetic acid solution, allowed to stand for 24 hours, and the lead content of the leachate determined.

In Canada, ceramics are regulated under the Hazardous Products Act. The ceramic and glazes regulation specifies an acid leachate test level of no more than 7 ppm lead. There is active enforcement of this regulation with field inspectors conducting sampling of glazed ceramics. Consumer and Corporate Affairs Canada is reviewing the 7 ppm limit with a view to reducing it in the future.

Most ceramics are found to be considerably below the leachate test limit because they are fired at high temperatures. However, articles which have been hand-painted on the inside and go through a second firing at a lower temperature (for example, in a wood-fired kiln) can exceed the acid leachate test limits. These products are usually not intended for use as food or drink containers (J. Gibson, personal communication). Consumer and Corporate Affairs is conducting a market survey of 200 to 300 items from different countries to estimate percentage failure of the acid leachate test on a broad, unbiased base. A relatively high percentage failure occurs for ceramics tested by inspectors because inspectors preferentially sample materials suspected of presenting a risk of lead exposure (J. Gibson, personal communication).

Crystal glasses and decanters may be another source of lead exposure due to the possibility of lead leaching into the beverage contained within. Lead, as lead oxide (PbO), is added to molten quartz to produce a glass that is high in density and durability with a brilliance not characteristic of normal glass (Graziano and Blum, 1991a). The quality of the crystal increases with the lead content; the latter can be as high as 32%.

Two recent studies show an increase in the lead content of wines and spirits after storage in glasses and decanters of varying lead content. The first study tested port wine stored in

three different types of decanters after 2, 7, 31, 84 and 127 days. After 127 days, the lead concentrations in the wine were found to be 5331, 3061, and 2162 µg/L (ppb) in decanters containing 32%, 32%, and 24% lead oxide, respectively (Graziano and Blum, 1991a).

The same researchers then tested 11 decanters containing different types of beverages with varying storage times. Beverages from 6 of the 11 had very high lead levels, ranging from 1.4 - 21.5 mg/L (Table C-23). Finally, four sets of four glasses containing 0%, 24%, and 32% lead oxide as well as an unknown content, were examined. A significant rise in the lead content of the wine was observed over a four hour period. The mean lead concentration in the 12 glasses rose from 33 µg/L at the beginning of the experiment to 68, 81, 92, and 99 µg/L after 1, 2, 3, and 4 hours, respectively (Graziano and Blum, 1991a).

In a separate analysis, the above researchers examined the extent of lead leaching into apple juice and infant formula from crystal baby bottles (Graziano *et al.*, 1991b). The lead concentration increased from 1 µg/L to 166 µg/L in four hours. In the infant formula, there was an immediate rise in concentration that was attributed to the elevated temperature of the liquid. The lead concentration ranged from 140-280 µg/L.

The elevated lead levels in wines and spirits have also been reported by other workers. The lead content was measured in various wines and spirits stored for 60 weeks in crystal with 20 - 30% lead oxide (Table C-24). All but one sample, Sample 4, surpassed the federal maximum allowable concentration of 200 µg/L (ppb). Sample 8, with the highest level of 2576 ppb, was the only sample showing a change in pH from 3.9 to 3.6. This suggests that acidic beverages promote the leaching of lead (Falcone, 1991). This pH dependency has been observed in wine by other workers (DeLeacy, 1987).

The Liquor Control Board of Ontario uses the standard acid leachate test for imported ceramic and crystal ware (4% acetic acid at room temperature for 18 hours) (Section C.3.3.3). The maximum permissible level of lead in the acid leachate is 7 ppm, but 200 ppb for alcoholic beverages. Thus, a vessel may be within guidelines while the alcoholic beverage within it may not pass the guidelines (Falcone, 1991).

TABLE C-23 LEAD CONCENTRATIONS OF BEVERAGES STORED IN CRYSTAL DECANTERS

BEVERAGE	STORAGE PERIOD (Approx.)	LEAD CONCENTRATION (µg/L)
Brandy	5 yr	7746
Madeira	5 yr	1402
Whisky	>3 yr	2587
Port	6-12 mo	203
Armagnac	6-12 mo	203
Brandy	>5 yr	19920
Brandy	>5 yr	21530
Brandy	5 yr	8390
Gin	6-12 mo	13
Brandy	>1 yr	68
Orange liqueur	>2 yr	173
Bourbon	18 mo	17
Tequila	18 mo	300
Vodka	1 yr	11

Source: Graziano and Blum, 1991a

TABLE C-24 LEAD CONTENT OF ALCOHOLIC BEVERAGES STORED IN LEAD CRYSTAL DECANTERS (ppb)

TIME (WKS)	SAMPLE 1 COGNAC (24%)*	SAMPLE 2 RYE (20%)*	SAMPLE 3 LIQUEUR (24%)*	SAMPLE 4 ARMAGNAC (24%)*	SAMPLE 5 SCOTCH (24%)*	SAMPLE 6 COGNAC (24%)*	SAMPLE 7 SCOTCH (30%)*	SAMPLE 8 PORT (24%)*	SAMPLE 9 COGNAC (20%)*
0	10	10	20	10	10	10	10	10	10
1	25	10	53	17	65	12	306	263	16
2	66	34	70	30	133	34	462	294	37
3	96	45	112	40	208	47	552	366	40
5	142	88	147	64	234	86	755	469	44
10	181	116	162	70	243	90	938	603	45
21	239	131	199	70	596	103	1168	894	78
34	403	181	262	105	1306	122	1573	1205	100
60	689	326	305	116	1586	239	1646	2576	169

* Lead oxide Source: Falcone, 1991

C.3.3.5**Cigarettes**

Airborne lead that settles on tobacco leaves before harvest can contaminate cigarettes. The lead content may be as high as 2.5-12.2 µg lead per cigarette. About 2-6% of the lead may be inhaled by the smoker (WHO, 1977, cited in Elias, 1985 and U.S.EPA, 1986; Harrison and Laxen, 1981). Secondhand smoke may contain 5-10% of the lead that was in the cigarette (Mahaffey, 1978).

C.3.3.6**Wine**

Wine may contain lead. European wine has an average range of 100 to 200 µg/L but may be as high as 300 µg/L. The average range for Californian wine is 100 to 300 µg/L (Elias, 1985; U.S.EPA, 1986). Recent data from the Liquor Control Board of Ontario shows that lead levels in European wines can be as low as 2.0 µg/L. The wines of the Americas can have lead levels as low as 2.6 µg/L (LCBO, 1991, unpublished data).

Until the early 1970's, lead foil caps were used on wine bottles. Corrosion of the caps could leave a residue on the lip of the bottle that could contaminate the wine during pouring. Most lead foil caps are now coated with tin plating or an acid-resistant substance.

C.3.3.7**Cosmetics**

The sale of cosmetics, either domestic or imported, is regulated by the federal Food and Drug Act. The latter governs the composition, safety, labelling, and advertising of cosmetics. Although lead is not specifically banned, the Act contains general provisions forbidding the sale of cosmetics which contain any substances injurious to the health of the user. Manufacturers and importers of cosmetics must file a list of ingredients, indicating the exact concentration or concentration range for each. However, this notification does not ensure that the cosmetic product meets the requirements of the Food and Drug Act. The responsibility for compliance rests with the manufacturer.

No information is available on the lead concentrations of cosmetics in Canada. There are historical reports in the literature of cosmetics containing lead compounds. Lead sulphide was discovered in a brand of mascara manufactured in India (Mahaffey, 1978). Lead acetate is one of the ingredients of dark hair-dye and the use of this product might result in some absorption of lead through the scalp (Mahaffey, 1978).

C.3.3.8**Bread Packaging**

The exterior printed labels on plastic bread packaging may contain lead-based pigments. In the United States, eighteen different bread bags were tested, representing 14 different

national brands from three chain supermarkets. Lead was detected in all but one bag (Weisel *et al.*, 1991).

Lead will not penetrate plastic so there is no exposure to lead from eating the bread packaged in such bags. However, some people invert the empty bags and reuse them, often to store other food. This practice allows direct contact of the painted label with the food. A survey of families with young children was carried out to determine the frequency of this practice.

The study found that 41% reused the bags, 22% turned them inside out and 16% inverted them and used them for food storage. Leaching of lead into the food is possible, especially if the food is acidic. A weak acid, such as vinegar, in contact with a lead pigment for as little as ten minutes could extract about 5% of the lead in the plastic (Weisel *et al.*, 1991).

C.3.3.9 Lead Shot

Based on the consumption of lead for the production of lead shot and on sales of the latter, it is estimated that approximately 1500 tonnes of spent lead shot enters the Canadian environment annually in the form of solid waste (Jaques, 1985a). In 1984, 47,000 tonnes of lead shot were used in the United States (U.S. Bureau of Mines, 1972-1984, cited in U.S.EPA, 1986).

The wide distribution of spent shot makes recovery of the lead virtually impossible. Lead shot remains in the environment, where it is frequently consumed by wildlife. It has been estimated that over one million waterfowl in North America die every year of lead poisoning as a result of consuming lead shot, or of embedded pellets. Exceptionally high quantities of lead shot may be found in the vicinity of firing ranges (DeMayo *et al.*, 1982).

Humans may also be exposed in this way. Cooking animals shot with lead pellets may release lead into the cooking medium and distribute it throughout the food. Also, if small enough, the shot itself may be consumed.

C.3.4 Drinking Water

Drinking water systems have traditionally used lead and lead-containing products because they are long lasting, easy to work with and readily available. The literature contains several reports of adverse health effects caused by lead in drinking water systems (Nriagu, 1985).

Most water mains in Ontario greater than 4 to 6 inches (10.2-16.2 cm) in diameter are made of cast iron, ductile iron, asbestos cement or, more recently, plastic. Smaller distribution lines (1-2 inches (2.5 cm-5.1 cm) in diameter) have been made of galvanized iron, copper, lead or plastic. Most small lead mains have been replaced. However, in some water distribution systems, the types of pipe are unknown, especially smaller sizes. As of the early 1980's there remained in metropolitan Toronto approximately 50000 lead mains. These mains are currently replaced by attrition.

Each house connected to the water system receives water through a service line, usually 0.5-0.75 inches (1.3-1.9 cm) in diameter. Until the 1950's, these were often made of lead because its flexibility allowed resistance to subsidence and frost heaving. Copper and, recently, plastic are now used, but many thousands of lead service lines are still in use, especially in cities.

House plumbing can also be a source of lead. Lead was previously used for all interior piping, but most of these old systems have been replaced by galvanized piping, copper or plastic pipes. Most new systems use copper piping joined with lead/tin solder. Lead-free solder is available: tin-antimony or tin-silver solder. However, there has been little incentive to use such products in home plumbing. A recent amendment to the Ontario Plumbing Code, which came into effect in March 1989, requires that solders and fluxes used in potable water systems must not contain more than 0.2% lead (Ont. Reg. 734, 1988). Another source of lead in drinking water may be the taps used in the home (Samuels and Meranger, 1984).

C.3.5 Agricultural Products

Agricultural products may act as a source of lead through the use of lead-containing pesticides or fertilizers.

C.3.5.1 Pesticides

In 1910, lead arsenate pesticides were recommended for use on Ontario fruit crops, because they did not damage crop foliage. Lead arsenate continued to be recommended for use on grape crops until 1942; vegetable crops until 1955; peaches until 1956; cherries until 1971; and apples until as late as 1975 (Frank *et al.*, 1976; Freedman and Hutchinson, 1981). Although this pesticide is no longer used on Ontario fruit crops, historic accumulation in the soil may present a problem. The lead content of the soil in some old orchard areas is still quite high.

C.3.5.2 Fertilizer

Sewage sludge, which may contain lead, is often used as an agricultural fertilizer. In the mid-1970's, almost half (41%) of the sewage sludge produced in Ontario was added to farmers' fields (Black and Schmidtke, 1974, cited in Frank *et al.*, 1976). Ontario has several guidelines for the application of sludge to agricultural land. Soil receiving sludge may have no more than 60 µg/g lead before application, and no more than 90 kg lead/ha may be added to the soil via sludge application (OMAF, MOEE, and OMOH, 1986). The high organic content of sludge may bind the lead, rendering it relatively unavailable to crops (RSC, 1986).

Lead is also present in commercial fertilizer. In the mid-1970's, fertilizer sold in Ontario contained 5-26 ppm of lead (Agriculture Canada, 1975, cited in Frank *et al.*, 1976).

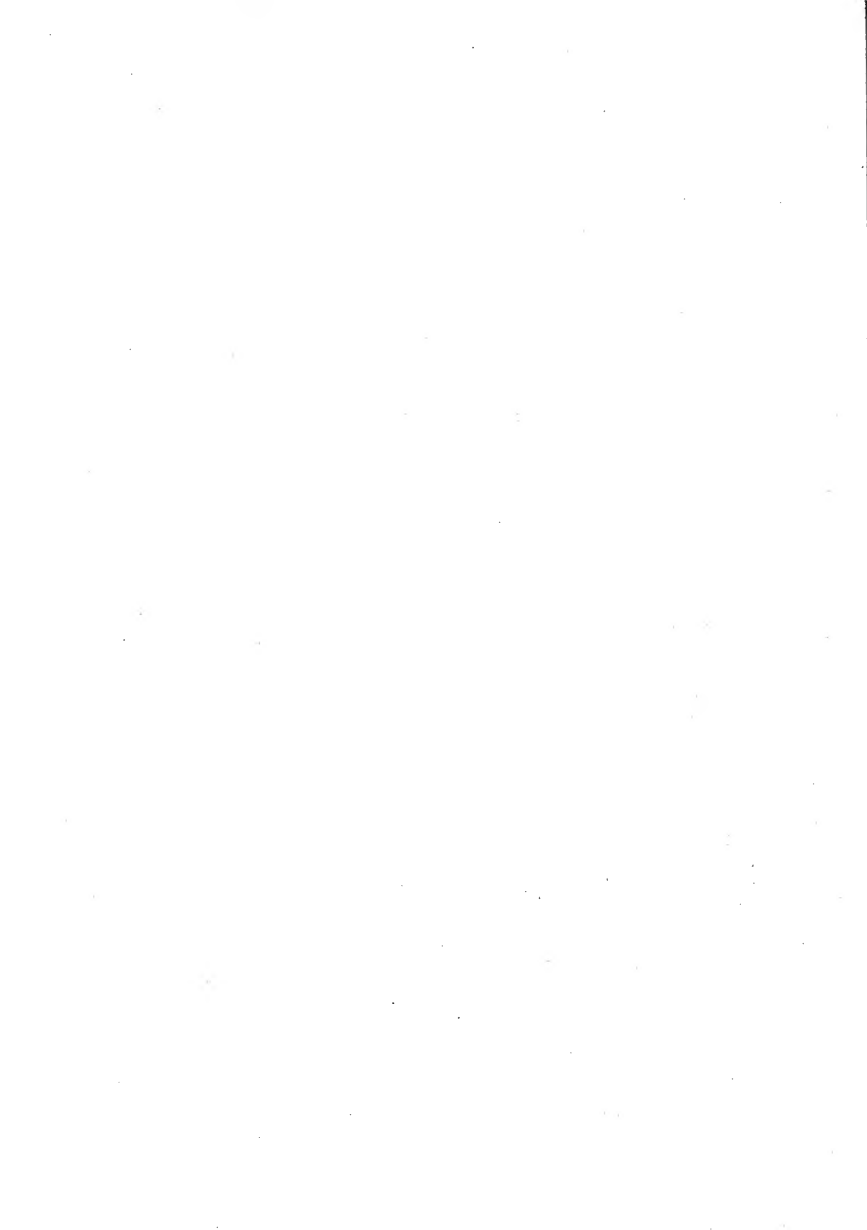
SUMMARY

- The Canadian environment is contaminated by lead almost entirely due to anthropogenic sources. In 1982, about 73,000 tonnes of lead were discharged into the Canadian environment with more than 83% in the form of solid waste. Atmospheric emissions and discharges into effluents accounted for nearly 16% and less than 1% respectively.
- In 1982, the total lead contained in solid wastes in Canada was estimated to be 61,000 tonnes which was six to seven times greater than the estimate for atmospheric emissions. Solid waste contributes the largest quantities, in large part from lead milling, smelting and refining industries.
- 1978 and 1982 inventories of sources and releases of lead to the Canadian atmosphere reported emissions to be 14534 tonnes and 11466 tonnes respectively. By 1987 lead emissions dropped to less than 6000 tonnes and the projection for 1995 is 4000 tonnes.
- The total Ontario lead emissions to the atmosphere were estimated to be 1504 tonnes in 1987, down from 3113 tonnes in 1982. The ten-year trend, 1980 to 1989, of ambient air lead levels in Ontario shows a significant decline. Presently, the most significant source of air pollution is the mining and milling of lead with total emissions to the Ontario atmosphere estimated to be 550 tonnes/year. The contribution from gasoline-powered motor vehicles has declined steadily with the phasing out of leaded gasoline. In 1982, lead emissions from gasoline accounted for 72% of lead emissions to the atmosphere whereas 1987 data shows leaded gasoline accounts for 43% of the total lead emitted in Ontario. Emissions from municipal solid waste incinerators are minor being estimated to emit about 25 times less lead than from all other known sources.
- Significant local impacts also occur in the vicinity of secondary lead smelters, three of which are located in Metropolitan Toronto. Together they constitute only about 0.5% of total lead emissions, but they are currently responsible for all of the measured exceedances of the 24 hour standard for particulate lead in air, and the 30 day point of impingement guidelines for lead in dust fall.
- Effluents from municipal sewage treatment and industrial activities also contaminate the environment, but to a smaller degree than atmospheric emissions and solid waste. The quantity of lead discharged in effluents represents only 5-6% of that emitted to the atmosphere. In 1982, Ontario discharged an estimated 125 tonnes of the total of 562 tonnes of lead in Canadian effluents. The largest source of lead in Ontario effluents, slightly more than two-thirds, was municipal sewage. In 1982, chemical manufacturing contributed 20% with the remaining 11% from smelting and refining, storage battery manufacture, primary iron and steel production, mining and milling, metal fabrication, foundries, refineries, and other miscellaneous industrial sources.

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- People and the environment are at risk of exposure from lead-containing consumer products including lead-based paints, ceramics, crystal, food cans, lead shot, wine, cosmetics, cigarettes, and bread packaging. In place are government regulations for lead-based paint and lead in ceramic ware.
 - Drinking water systems have traditionally used lead plumbing. New systems use lead-free alternatives, but many distribution systems still use lead service lines. Many older dwellings still contain lead indoor plumbing.
 - Agricultural pesticides no longer contain lead, but lead concentrations are still elevated in the soil of some Ontario orchards due to the historic application of lead arsenate. Lead may still be applied to crops in the form of sewage sludge and commercial fertilizer.

APPENDIX D

**ENVIRONMENTAL CHEMISTRY OF LEAD:
FATE, LEVELS AND TRENDS**



APPENDIX D

ENVIRONMENTAL CHEMISTRY OF LEAD: FATE, LEVELS AND TRENDS

It appears that the lead concentrations in the Great Lakes have declined since the 1970's. The major loadings of lead into the Great Lakes appear to be due to atmospheric deposition. Based on a mass-balance calculation of historical data up to 1985-1986 estimated atmospheric lead loading to Lakes Superior, Michigan, Huron, Erie and Ontario are 97%, 99.5%, 94%, 39% and 50% respectively. The greatest lead input was to Lake Erie reported at 567×10^3 kg per year.

Most of the lead is trapped in sediment where concentrations of lead also appear to have declined. Elevated lead levels in both water and sediment have been measured near industrial point sources. Concentrations of lead in 448 locations in and adjacent to the Great Lakes region was found to range from 5 $\mu\text{g/g}$ to 20000 $\mu\text{g/g}$.

The concentration of lead in aquatic biota are also decreasing. The sharpest decline has been seen at Maitland, located in the St. Lawrence, where the now closed alkyllead production plant was located. In 1981, alkyllead in whole carp samples had measured alkyllead levels of 4.207 ppm lead in comparison to 0.120 ppm in 1987. Benthic organisms have not seen as sharp a decrease due to the persistence of lead in sediment.

The major source of soil lead is atmospheric deposition. Lead tends to be retained strongly in soil, with little tendency to migrate into groundwater. Because lead is ubiquitous in nature, even soils in remote rural areas will have some quantity of lead. Generally, rural levels in Ontario are below 50 ppm and urban areas about an order of magnitude higher. The Ontario Ministry of the Environment has set 500 ppm as the upper limit of normal for soil lead in urban areas. Considerably higher levels may occur near motorways, houses with peeling paint or industrial point sources. Terrestrial animals do not appear to present an important route of human exposure. Terrestrial plants, however, reflect soil lead levels and lead tends to accumulate in the roots. Consumption of vegetables from urban gardens with elevated soil lead may present a significant source of human exposure.

INTRODUCTION

Exposure to lead may be direct or indirect. In indirect exposure, lead reaches humans through different environmental pathways: through atmospheric transport, water systems, or the food chain. Knowledge of the levels of lead in the environmental media along these pathways is essential for a comprehensive exposure assessment.

The transport and eventual fate of lead in the environment is influenced by its interaction with a given environmental medium, this interaction depending on the physicochemical properties of both lead and the medium. This chapter describes the physical and chemical properties of lead and its fate in the environment. Its levels in various media are reported: levels in air, soil, water, and non-human biota. Wherever possible, Ontario data are used. Reliance has been placed on published primary sources.

The term "lead" as used in this chapter refers to inorganic lead unless specifically named as organolead. Organolead compounds are considered to be critical pollutants and are discussed in Appendix E.

There is a continuous transfer of lead between air, water and soil. When modelling the fate of lead in the environment, the physical and chemical properties of lead must be considered as well as media-specific and site-specific parameters.

A more predictive method of modelling the movement of a given substance in the environment is the mass balance approach, which considers partitioning, transport and reaction processes. It is necessary to know the concentrations of a given substance in the various media as well as its concentrations in the input and output sources. A complete data set for a substance often does not exist, making the development of a mass balance construct difficult.

Although the data for lead are not complete to allow for a thorough cycling analysis, considerable information is available, so that the projection of trends within individual media is possible. The *aquivalence model* was developed to predict the fate of lead, and other involatile chemicals, in aquatic environments (MacKay and Diamond, 1989). It has been used to model the transport of lead in Lake Ontario.

The International Joint Commission on the Great Lakes has studied the role of atmospheric deposition on the mass balancing of toxic chemicals in the Great Lakes by calculating inputs and outputs (Strachan and Eisenreich, 1988). However, the models predicting the fate of lead in aquatic environments have not been linked to those for atmospheric environments. Individual media models, such as the *aquivalence model* and input-output calculations, have been used in an attempt to determine the whole lead cycle. This has proved to be difficult because of the many possible sources of lead and because often the source, usually anthropogenic, is not known. There are many data on the actual levels of lead in various environmental media but a lack of knowledge on the sources of lead and the routes it follows.

APPENDIX D-1

LEAD IN THE ATMOSPHERIC ENVIRONMENT

APPENDIX D-1 LEAD IN THE ATMOSPHERIC ENVIRONMENT

D-1.1 Fate of Lead in the Atmosphere

Soil and sediments are the two primary sinks for lead deposition through the atmosphere. There are two types of deposition: wet (rain or snow) and dry deposition, with the average residence time ranging from 7-30 days (ATSDR, 1988).

The gas-particle partitioning of a substance is dependent on the particle size and surface area, the organic content of the aerosol and temperature. The less volatile the substance, the greater its affinity for suspended particles. Lead's partitioning in the atmosphere is skewed toward the particulate phase due to the metal's relative involatility. The chemical form of the lead bound to suspended matter may vary, depending on atmospheric conditions and the nature of the suspended matter.

For the wet deposition process, the scavenging coefficients for both the gas and particle phases have been determined, based on temperature, the gas constant and Henry's Law constant (Eisenreich and Strachan, 1989). The Henry's Law constant for lead is negligible due to its low volatility. Thus, the total scavenging capacity is based on the particulate phase. The deposition and transformation of lead in air will depend both on the characteristics of the particulate and on the chemical properties of the scavenging precipitation. In other words, the chemical and physical properties of the rain could cause a change in the properties of the lead particulate. Rain can also influence the amount of lead particulate removed from the air. For example, where acid rain is prevalent, greater amounts of lead are expected to be removed by wet deposition because acid rain tends to solubilize lead.

Dry deposition is highly dependent on particle size and the degree of atmospheric turbulence. Lead particles greater than two microns (2 μm) in diameter settle out rapidly close to the source of emission. Smaller particles, however, can be transported many kilometres from the source depending on the wind. The flux of particles to a surface can be expressed as the product of the deposition velocity, at a given reference height, and the chemical concentration of the particle phase in the atmosphere (Eisenreich and Strachan, 1989). Such small size particles will travel further and contribute more to the wet deposition of lead. Dry deposition measurements have been made at sites remote from lead sources, where the long range transport would be the major contribution.

The International Joint Commission on the Great Lakes (IJC) has estimated the atmospheric loading of lead to the Great Lakes (Strachan and Eisenreich, 1988). The following parameters were considered: atmospheric and precipitation concentrations; mass transfer coefficients; and physical speciation in the atmosphere and in water. There is some uncertainty in the estimates because of the contribution of non-atmospheric lead sources and the difficulty in isolating these. The estimated lead loadings to the Great Lakes are presented in Table D-1-1. The fractions attributed to atmospheric pathways are indicated.

TABLE D-1-1 ESTIMATES OF ATMOSPHERIC LEAD LOADINGS TO THE GREAT LAKES

	*TOTAL INPUTS 10 ³ kg yr ⁻¹	% ATMOSPHERIC	
		Direct	Indirect
Lake Superior	241	97	0
Lake Michigan	543	99.5	0
Lake Huron	430	94	4
Lake Erie	567	39	7
Lake Ontario	426	50	23

- Adapted from Strachan and Eisenreich, 1988; Intermedia Pollutant Transport, 1989
- * Speciation not known therefore reported as total lead

The total atmospheric deposition was estimated to be the total of the lead which falls on the lake surface (direct) and the lead which falls "upstream" and flows through the connecting channels to the "downstream" lake (indirect) (Strachan and Eisenreich, 1988). The larger lakes, as is evident, receive a greater input from atmospheric sources due to their larger surface areas.

The input-output calculations for lead in the Great Lakes are given in Table D-1-2. The net flux of lead was outward for all the lakes except Lake Michigan (Eisenreich and Strachan, 1989; Strachan and Eisenreich, 1988).

Atmospheric deposition to soils is not well documented. Both wet and dry deposition occur with the same transformation scenario as the air-water interface. Urban centres where lead smelters are located will tend to have higher soil lead concentrations than rural areas. Areas close to the industrial site will have the highest levels, indicating large particle deposition close the source.

TABLE D-1-2 INPUT-OUTPUT CALCULATIONS FOR THE GREAT LAKES (KG/YR)

LAKE REGION	INPUT	OUTPUT	NET FLUX
Lake Superior	241	828	-587
Lake Michigan	543	472	71
Lake Huron	430	496	-66
Lake Erie	567	2010	-1440
Lake Ontario	426	490	-64

Adapted from Strachan and Eisenreich, 1988; Eisenreich and Strachan, 1989

D-1.2 Levels of Lead in the Atmosphere

D-1.2.1 Levels of Lead in Ambient Air

Lead levels well in excess of the $5 \mu\text{g}/\text{m}^3$ standard have been measured in areas with extremely heavy traffic or under adverse meteorological conditions, or in enclosed areas like garages. These readings occurred outside Ontario (NRC, 1978). Similar levels could possibly occur in Ontario, but have not been detected through routine monitoring.

Ambient lead levels have dropped significantly since 1970. During the 1980's, in particular, mean monitored levels in Ontario dropped from 0.3 to $0.01 \mu\text{g}/\text{m}^3$ (Figure D-1-1) (MOEE, 1991d). It is noted, however, that monitoring is concentrated in urban and industrialized areas, or in areas with heavy motor vehicle traffic. The levels in rural areas may be much lower. In the mid-1980's, most rural sites had an annual mean of $0.1 \mu\text{g}/\text{m}^3$ or less (MOEE, 1986a; 1987d; 1988c; 1988d; 1988e). Recent monitoring data indicate a further drop in mean lead concentrations in rural areas to $0.01 \mu\text{g}/\text{m}^3$ (P. Kiely, personal communication, 1991). The analytical detection limit for lead in air is $0.01 \mu\text{g}/\text{m}^3$ (high volume sampler) so that actual concentrations could be even lower.

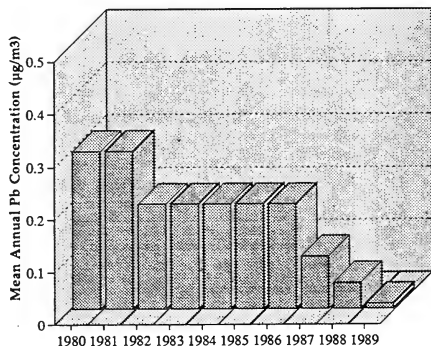


Figure D-1-1 Ambient Air Lead Levels, 1980-1989

The only recent exceedances of the 24-hour ambient air quality guideline have occurred at sites within 100 metres of one of the three secondary lead plants in Ontario: Canada Metal Company; Toronto Refiners and Smelters Ltd.; and Tonolli Company of Canada Ltd. The highest measured one-hour value was $40.3 \mu\text{g}/\text{m}^3$. It was found in 1985 at a sampler on Tecumseh Street, near Toronto Refiners and Smelters Ltd. (MOEE, 1986a; 1987d; 1988c; 1988d; 1988e). In 1989, monitoring stations near the Canada Metal Company and Tonolli Ltd. reported the number of exceedances of the 24-hour air quality criterion as two and 23, respectively (MOEE, 1991d). In 1988, the reported number of exceedances increased to three for the Canada Metal Company and 64 for Tonolli Ltd. (MOEE, 1990a). Since that time, the exceedances have decreased in frequency and concentrations. The Toronto Refiners and Smelters Ltd. is now closed.

Secondary lead smelters have a much greater impact than that implied by their relative contribution to total lead emissions. Their lead emissions are mechanically generated at or near ground level by material transport and handling, and to a much lesser extent, by resuspension of soil. The lead concentrations, however, fall off rapidly with increasing distance from the source.

D-1.2.2 Levels of Lead in Dustfall (Dry Deposition)

Ontario has a "point of impingement guideline" for dustfall set at $0.1 \text{ g}/\text{m}^2/30 \text{ days}$ ($100000 \mu\text{g}/\text{m}^2/30 \text{ day}$). Since 1980, the levels of lead in dustfall have generally remained below 10% of this guideline at virtually all dustfall stations in Ontario. Values at rural sites remote from roadway contributions are below 5% of this figure.

Several studies have found a relatively high proportion of suspended lead particulate in the submicron range. Values ranged from $0.0005 \text{ g}/\text{m}^2/30 \text{ days}$ ($500 \mu\text{g}/\text{m}^2/30 \text{ days}$) in southern Ontario to $0.0001 \text{ g}/\text{m}^2/30 \text{ days}$ ($100 \mu\text{g}/\text{m}^2/30 \text{ days}$) in northern Ontario (Reid *et al.*, 1988a). These measurements fall well below the Ontario lead in dustfall guideline of $0.1 \text{ g}/\text{m}^2/30 \text{ days}$ ($100000 \mu\text{g}/\text{m}^2/30 \text{ days}$).

The exceptions are those sites in close proximity to one of the three secondary lead smelters, where violations of the guideline, based on 1987 data, have been frequent. The highest reading in dustfall ($0.451 \text{ g}/\text{m}^2$ or $451000 \mu\text{g}/\text{m}^2$) occurred in April, 1978 at a site close to the Tonolli Company of Canada Ltd. (MOEE, 1988d; 1988e). Elevated levels appear to persist as far as 500 metres from each of the smelter complexes, beyond which they fall to typical urban background levels of about $0.01 \text{ g}/\text{m}^2/30 \text{ days}$ ($10000 \mu\text{g}/\text{m}^2/30 \text{ days}$) (MOEE, 1988d; 1988e).

D-1.2.3 Levels of Lead in Precipitation (Wet Deposition)

The Ontario Precipitation Monitoring Network measures lead concentrations in precipitation. Values range from approximately 8 µg/L in the south to 3 µg/L in the north (Reid *et al.*, 1988a; 1988b). Values would be expected to be higher in urban areas, particularly in Toronto, due to heavy automotive traffic and other local sources of lead. The Precipitation Network, however, does not include sites in the Metropolitan Toronto area. An average lead concentration of 50 µg/L was reported for Toronto rain in 1973 (NRC, 1978). Present levels would be expected to be less than one-third of this value as observed for the reduced air concentrations.

Wet deposition of lead in Ontario ranges from 0.0006 µg/m²/30 days in the south to 0.0002 µg/m²/30 days in the north. Contributions to total lead deposition by wet and dry deposition are similar in magnitude although the contribution from wet deposition is slightly greater. Deposition trends over the past five years have declined considerably, reflecting the significant drop in automotive lead emissions in this part of the continent (Reid *et al.*, 1988a).

APPENDIX D-2

LEAD IN THE AQUATIC ENVIRONMENT

APPENDIX D-2 LEAD IN THE AQUATIC ENVIRONMENT

D-2.1 Fate of Lead in the Aquatic Environment

Lead may be present in the aquatic medium as a result of natural causes; direct deposition or dumping; or atmospheric deposition. The major loadings of lead into Lake Ontario have been determined to be atmospheric deposition (wet and dry) and inflowing suspended particles (MacKay and Diamond, 1989).

Lead has a low vapour pressure and thus, once deposited in water, does not volatilize into the atmosphere. Lead exists in the aquatic medium in three phases: dissolved lead, suspended particulate and sediment. Most of the lead in water is lost to sediment. This flux is due to a balance between sediment deposition/resuspension and diffusional flow, the latter being relatively insignificant.

Lead is slightly soluble in water. Thus, there is the potential for human exposure through drinking water. The degree to which water is corrosive to lead in plumbing materials depends on several factors, particularly the pH and alkalinity of the water. In pure water, the $Pb(II)$ cation is partially hydrolysed to the hydroxide, $Pb(OH)_2$. In natural water, this reaction is largely prevented by the presence of sulphates and carbonates, which form a protective coating on the surface of lead. This coating may form on the inner surfaces of lead pipes used for drinking water, thereby decreasing the leaching of lead into the water. The degree to which this may occur is highly variable and is a function of the sulphate and carbonate concentrations.

Lead that remains dissolved in the water is available for uptake by biota. Lead does not biomagnify, rather it biominifies. Biominification is advantageous to biota because less travels up the food chain. Nevertheless, an extremely high level in water would mean moderate levels in fish that could be toxic to the fish or to those who consume it.

Lead in water will partition between the dissolved phase and the particle phase. The ratio of suspended lead particulate to dissolved lead has been found to vary from 4:1 in rural streams to 27:1 in urban streams (U.S.EPA 1986a, as cited by ATSDR, 1989). The degree of partitioning is believed to be dependent on the concentration of suspended particles. As the settling suspended particles increase in concentration in the sediments, their affinity for lead drops. Upon resuspension, the affinity for lead increases due to a decrease in particulate concentration in the sediment (MacKay and Diamond, 1989). The movement of lead may thus be dictated by the concentration of suspended particles; by their chemical composition; or by some other factor related to the position of the particles in the lake.

Factors affecting sediments, either their bulk movement or their capacity for lead, have the greatest influence on lead concentrations in water and biota (Clark and Diamond, 1988). If the sediment can hold more lead, then less lead will be present in the water and less will be

available for uptake by the food chain. The affinity of sediment for lead can be altered by increasing the organic content and the iron oxyhydroxide or by shifting the particle size spectrum to include fine particulate and clays (Appendix E of Clark and Diamond, 1988). The sedimentation process may also vary with the seasons or with differing climatic changes. For example, an increase in algae in a particular season can have a positive effect on sedimentation.

Lead will remain in the sediment compartment unless resuspension occurs. Resuspension may be promoted by water turbulence which releases lead back into the water column in the form of suspended particulate. Ideally, once particulate has sedimented to the bottom it should become buried, a process which removes lead from the water column. This process may be influenced by ice cover which decreases water turbulence. The rates of sedimentation and resuspension in a given body of water would contain some uncertainty and variability due to the dynamic nature of lakes. For Lake Ontario, a sediment deposition of 88% has been reported for outflow. Of the sedimented portion, 23.5% became resuspended and 12% was buried (MacKay and Diamond, 1989).

The equivalence model was used to model the transport of lead in Lake Ontario (MacKay and Diamond, 1989). The model assumed prolonged, constant lead emissions and air concentrations. Simple algebraic equations were used to capture the dominant processes (steady state). The equivalent concentration was defined for all phases. It is linearly related to the actual concentration through a proportionality constant equal to the phase-to-water partition coefficient (MacKay and Diamond, 1989). The model also incorporated transport and transformation parameters to account for the rates of different processes. The model accurately predicted the concentrations of lead in air, rain and sediment but underestimated the concentration in water.

Atmospheric deposition plays a major role in loading lead into aquatic environments. The estimated loadings to Lake Ontario occur through three major routes: direct emissions, inflow and air-water exchange. In 1985, the loadings were 1752 kg (0.4%), 210,073 kg (45%), and 256,656 kg (55%), respectively (MacKay and Diamond, 1989). Although the inflow is from the tributaries, the concentration of lead probably arises primarily from atmospheric deposition to the tributaries. The various process rates of lead in Lake Ontario are illustrated in Figure D-2-1 (MacKay and Diamond, 1989).

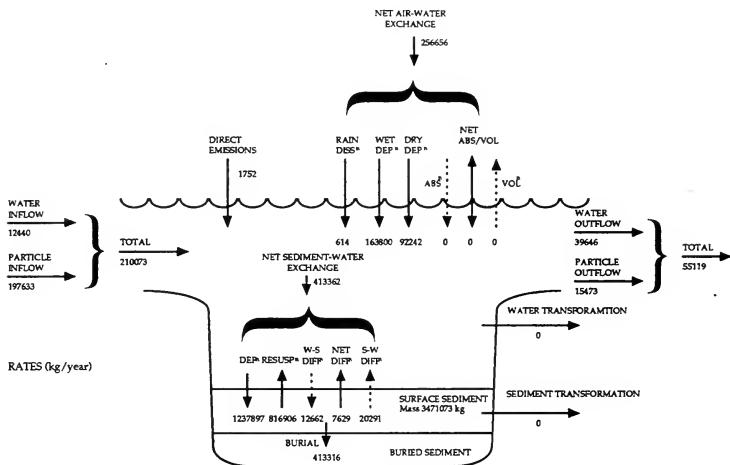


Figure D-2-1 Process Rates and Masses of Lead in Lake Ontario Under Steady-State Conditions (Source: Mackay and Diamond, 1989)

D-2.2 Levels of Lead in the Aquatic Environment

Lead may be present in the aquatic medium in three separate forms: dissolved, as suspended particulate or in sediment. Lead is most commonly measured as total lead, which consists of the inorganic and organic lead present in solution and in the particulate form. Suspended particulate eventually deposits in sediment so that the lead content of sediments is dependent upon the amount present as particulate.

Lead is available to aquatic life through all three forms. Phytoplankton and zooplankton are exposed to both dissolved and suspended lead. Benthic species, both flora and fauna, would have access to the lead present in the sediments.

In 1986, the International Joint Commission of the Great Lakes (IJC) identified lead as a critical pollutant for its Primary Track. Considerable information is available on the levels of lead present in Ontario waters. Data are presented in this section on the lead levels found in major bodies of inland waters.

Comprehensive surveys have been completed that allow for spatial and temporal determinations as well as inter- and intra-lake comparisons (Rossman, 1982, 1983, 1984, 1986; Rossman and Barres, 1988; Strachan and Eisenreich, 1988). Wherever possible, the total lead concentrations are presented to allow for comparisons between phases.

Monitoring data from four Ontario drinking water surveys are also discussed to allow an assessment of the lead consumed in drinking water.

D-2.2.1 Surface Waters

D-2.2.1.1 Dissolved Lead

Some of the historical data on the levels of dissolved metals in the water from open lakes has been questioned. Sample contamination may have occurred due to its presence on platforms and in laboratory dust (Environment Canada, 1991). Recent studies often report significantly lower lead levels. This may be due to a true decrease in lead contamination, to improved analytical techniques or to both factors. Background concentrations of total lead have been reported to range between 0 to 10 µg/L.

Chemical mass balance calculations have shown that atmospheric deposition is the major process by which contaminants such as lead are introduced to Lake Superior (Strachan and Eisenreich, 1988). In general, the concentrations of dissolved or total lead in Lake Superior were either significantly less than or similar to concentrations in the other Great Lakes. Lake Ontario has significantly lower levels of dissolved lead than Lake Michigan and significantly less total lead than Lake Erie (Rossmann and Barres, 1988). Specific findings in each of the Great Lakes are discussed below.

It has not been possible to determine a temporal trend in the lead levels of *Lake Superior*, upon comparing 1983 data to the historical data on contaminants in dissolved, particulate and total water. In the 1980's, however, high levels of lead, in dissolved and total water, were reported in certain harbours and bays (Simpson, 1987; MOEE, 1986b; Glass *et al.*, 1988).

Lake Michigan lies completely within United States territory, but must be considered because of its obvious relationship with the other Great Lakes and for inter-lake comparisons. The levels of dissolved lead in Lake Michigan have been reported in three studies: two analyzed data from 1981 (Rossmann, 1984; Rossmann and Barres, 1988) and the third determined a mean lead value based on historical data up to 1985-1986 (Strachan and Eisenreich, 1988). The mean value reported in these studies was 150 ppt. The mean total lead concentrations, however, have been reported to be 7000 ppt prior to 1981 and 260 ppt, post 1981 (Rossmann,

1984; Rossmann and Barres, 1988). The 7000 ppt level was probably elevated due to sample contamination.

The lead concentrations of *Lake Huron* have been reviewed. Comparison of 1980 data to those from previous studies demonstrated a downward trend in dissolved lead (Rossmann, 1983). In 1980, the dissolved lead concentration was reported to be 19 ppt (Rossmann, 1982; Rossmann and Barres, 1988). A mass balance calculation on historical data up to 1985-1986 found the dissolved concentration to be 150 ppt (Strachan and Eisenreich, 1988). The mean total lead was determined to be 38 ppt (Rossmann, 1982; Rossmann and Barres, 1988).

A review of 1981 monitoring data on dissolved lead levels in *Lake Erie* found mean concentrations to be 220 ppt (Rossmann, 1984; Rossmann and Barres, 1988). This does not agree with the historical mass balance analysis which reported a concentration of 750 ppt (Strachan and Eisenreich, 1988). The data on the total water lead concentration are inconsistent, with levels ranging from 150 ppt to 26310 ppt (Rossmann, 1984; Rossmann and Barres, 1988). Again, the elevated values may be due to sample contamination.

Lead levels in *Lake Ontario* are consistently low, with higher levels recorded in a small westerly zone of the lake (Schmidt and Andren, 1984; Neilson, 1983). The mean dissolved lead concentration has been determined to be 300 ppt (Strachan and Eisenreich, 1988). The mean total lead concentration, as reported by various investigators from 1979 to 1985, ranges from 100 ppt to less than 500 ppt (Neilson, 1983; Stevens, 1987; Rossmann and Barres, 1988).

The *Niagara River* connects Lake Erie to Lake Ontario and has several industrial point sources of contamination along its banks. In 1983, data on the contamination of the Niagara River were published (Allan *et al.*, 1983). Since then, many papers have appeared and a National Toxics Committee study has been conducted (NRTC, 1984). Surveys were carried out at Fort Erie and Niagara-on-the-Lake in 1985 and in 1986-1987. The mean total lead concentrations found at Fort Erie were 1000 ppt at both times. At Niagara-on-the-Lake, they were 1400 ppt and 1600 ppt, respectively (Kuntz, 1988b; DOE/MOEE, 1988). The Strachan and Eisenreich analysis concurred with the level of 1000 ppt (Strachan and Eisenreich, 1988).

Major industries are situated along the *St. Lawrence River*. A tetraethyl lead (organic lead) plant, located near Maitland, has discharged tetraethyl lead into the St. Lawrence. The level of lead in the water in 1977 was 1.01 ppb based on unfiltered water (Chan, 1980). Between 1977 and 1983, the mean concentration of inorganic lead was 1.2 ppb (Sylvestre *et al.*, 1987). The current levels are expected to be much lower due to the closure of the production plant. The *St. Clair River* drains Lake Huron and flows into Lake St. Clair. Several petrochemical plants and refineries discharge into the river so that industrial discharges exceed municipal discharges (Marsalek, 1986). The point sources have resulted in high levels of contaminants in the water and sediment.

Concentrations of lead in water are generally below the objectives set for the Great Lakes (GLWQA) except near Sarnia and Corunna. Near Corunna, an alkyl lead production plant

has contributed to the elevated lead levels. Typical lead water levels range from 10 to 100 ppt and can be as high as 2.7 ppb (unfiltered water) in industrialized areas (UGLCCS, 1989).

Filtered total lead levels ranging from 27 to 103 µg/L have been measured in some lakes in the Sudbury region (Stokes *et al.*, 1973).

D-2.2.1.2 Suspended Particulate

The mean concentration of lead in suspended particulate from *Lake Superior* was measured at 770 and 25 in 1977 and 1983, respectively. A mean mass balance analysis of historical data up to 1985-1986 determined the level to be 25 ppt (Eisenreich, 1982; Rossman, 1986; Rossman and Barres, 1988; Strachan and Eisenreich, 1988). Based on the 1983 data, as for dissolved lead, mean concentrations of lead in suspended particulate were significantly less or not significantly different from the findings in the other Great Lakes (Rossman and Barres, 1988).

A historical survey was carried out by Strachan and Eisenreich (Strachan and Eisenreich, 1988) on the measured concentrations of suspended lead in *Lake Michigan* up to 1985-1986 and determined the mean concentration to be 50 ppt.

In 1980, a survey was conducted measuring the toxic metal concentrations in the suspended particulate of *Lake Huron* (Rossman, 1982; Rossman and Barres, 1988). The measured value was 50 ppt, which concurred with the mass balance analysis conducted by Strachan and Eisenreich (Strachan and Eisenreich, 1988).

Lake Erie has been heavily contaminated with metals due to the presence of industry. Strachan and Eisenreich through their mass balance approach, determined the mean lead concentration in suspended particulate to be 250 ppt (Strachan and Eisenreich, 1988). A median value of 190 ppt has also been reported (Rossmann and Barres, 1988).

Lake Ontario has also been contaminated due to the close proximity of many anthropogenic sources. In 1978 and 1985, approximately two-thirds of the lead in the lake was in the suspended particulate form (Nriagu *et al.*, 1980; Rossmann and Barres, 1988). The mass balance calculation for suspended matter determined the lead concentration to be 100 ppt (Strachan and Eisenreich, 1988).

In 1978 and 1979-1980, Environment Canada and the Ontario Ministry of the Environment carried out studies on suspended sediments in the Niagara River at locations between Fort Erie and Niagara-on-the-Lake. The mean lead concentrations in the suspended fraction were found to be 80 and 58 ppm, respectively (DOE and MOEE, 1981). Two surveys carried out in 1981 and 1982 found the mean concentration for both years to be 93 ppm (Kuntz, 1984).

The suspended sediment fraction of the *St. Clair River* is very large due to the erosion of Lake Huron. The shallow nature of the river leads to the resuspension of bottom sediments.

The role of sediments as a source of chemical contaminants to the aquatic environment is not well understood. Because lead is ubiquitous in the environment, some level of lead would be expected to be detected in sediments. Background ranges of lead in the depositional basins of the Great Lakes are presented in Table D-2-1. These concentrations are representative of pre-colonial sediments, as determined by the Ambrosia pollen horizon or by radionuclide dating at the time of testing (Mudroch *et al.*, 1988). Reported background levels in nondepositional zones, embayments and harbours are also included in the table.

TABLE D-2-1 BACKGROUND LEAD LEVELS IN SEDIMENTS OF THE GREAT LAKES

	LAKE ONTARIO	LAKE ERIE	LAKE HURON	LAKE MICHIGAN	LAKE SUPERIOR
Depositional Basins	18-32	21-49	14.4-36	8-10	20.5-68.01
Nondepositional Zones	25	-	-	-	18.4-24.6
Embayments	-	-	83.9-93.0	8-29	-
Harbours	28	-	-	-	-

Adapted from Mudroch *et al.*, 1988

A guideline has been developed for determining appropriate disposal techniques for dredged materials. In Ontario, this guideline, called the Open Lake Disposal of Dredged Materials Guideline, has been set at 50 ppm.

Sediment quality guidelines for lead have been drafted based on the study of 448 locations and 95 different species in and adjacent to the Great Lakes region. Sediment concentrations ranged from 5 µg/g to 20000 µg/g. The guideline of 31 µg/g is based on the lowest effect level, defined as the 5th percentile of the screening level concentrations of the organisms studied. In other words, the majority of benthic organisms are believed to be unaffected at this level. A severe effect level at 250 µg/g, was defined at the 95th percentile of the screening level concentration (MOEE, 1990d draft).

A multi-year study has been done, mapping the distribution of chemicals in the surficial sediments (3 cm depth) of the five Great Lakes (Thomas and Mudroch, 1979). The concentrations and distributions of metals were principally related to the geology of the area surrounding the lake.

The lead concentration in the surface sediment of *Lake Superior* normally measured less than 100 ppm (Kemp *et al.*, 1978; Strachan and Eisenreich, 1988; IJC, 1977c). The historical trends in loading have been determined by collecting, sectioning, age dating and analyzing the lake

sediment cores. Lead loading to Lake Superior has increased steadily from the early 1900's to the present, peaking in the early 1970's (Rossmann, 1986). Since that time, loading has declined, as observed in the other Great Lakes.

In 1975, an extensive superficial sediment survey was done in *Lake Michigan*. The mean lead concentration was found to be 40 ppm (Cahill, 1981). This concurs with the findings of two other investigations (Strachan and Eisenreich, 1988). The distribution of lead in the sediment cores of Lake Michigan has decreased to levels equivalent to 1950 and 1960 levels, suggesting a decline in lead loading (Goldberg *et al.*, 1981).

The results from various studies of the superficial sediments of *Lake Huron* are not in agreement. Earlier studies reported the lead concentrations to be less than 100 ppm (Thomas, 1973; U.S.EPA, 1980). Later findings set the surficial lead concentration at 151 ppm with a higher concentration of 230 ppm in specific embayments (Mudroch *et al.*, 1988).

Studies were done in 1971 and 1979 on the lead concentration in the bottom sediment of *Lake Erie* (Frank *et al.*, 1977; Thomas and Mudroch, 1979; Rathke, 1984). A comparison between the two studies is not possible because the 1971 study used 3 cm samples while the 1979 study used 10 cm samples. A mean lead concentration of 87 ppm was found in 1971 and range of 50-140 ppm in 1979.

The levels of lead in *Lake Ontario* bottom sediments are greater than those of the other Great Lakes. In 1968, the mean lead concentration in the bottom sediments of Lake Ontario was reported to be 107 ppm at 3 cm (Thomas and Mudroch, 1979) and 220 ppm at 1 cm (Kemp and Thomas, 1976). Analysis of historical data up to 1985-1986 determined the lead concentration to be 100 ppm (Strachan and Eisenreich, 1988). Other studies have determined the range of concentrations to be 50-150 ppm (Sonzogni and Simmons, 1981 as cited in Environment Canada 1991) and 7-285 ppm (Mudroch *et al.*, 1988).

Bottom sediment samples at various Canadian and American locations along the *St. Lawrence River* have ranged in lead concentration from less than 50 ppm to 190 ppm (Kuntz, 1988a). The levels are expected to decline due to the closure of the alkyl lead production plant at Maitland.

The bottom sediment samples from the *Niagara River* are not comparable to those taken from depositional basins. The former samples are representative of the relative point sources close to the sample location. A mean lead concentration of 98 ppm was reported in 1981 at a depth of 2.54 cm (Kuntz, 1984). Environment Canada and the Ontario Ministry of Environment conducted two studies in 1979, reporting ranges of 4-200 ppm and 6-60 ppm (DOE and MOEE, 1981).

Lead levels in the bottom sediment of the *St. Clair River* and *Lake St. Clair* are normally low except in specific areas. Elevated levels have been reported at Corunna (339 ppm), Sarnia, Black River and the eastern area of the St. Clair Delta (UGLCCS, 1989; Mudroch *et al.*, 1985a,

1985b). The elevated levels are indicative of the large degree of industrialization near the Detroit River.

Lead levels in the bottom sediment of the *Detroit River* often exceed the dredge spoil guideline of 50 ppm. In 1983, the levels were greater than those of both Lake St. Clair and Lake Erie (peak concentration, 700 ppm) (Mudroch, 1985a, 1985b). Other elevated concentrations have been reported as follows: 960 ppm in 1982 (Lum and Gammon, 1985); 704 ppm and 546 ppm in 1983 (Mudroch, 1985a, 1985b; Chau *et al.*, 1985); and 1750 ppm in 1982 and 1985 (UGLCCS, 1989).

Data on bulk sediment lead concentrations for the years 1983 through to 1987 are presented in Table D-2-2 to illustrate the ranges in lead concentration. Sampling locations lie within designated areas of concern within the Great Lakes region and have been selected for remedial action by the International Joint Commission. The sediment quality of these areas is continually monitored. For example, the St. Lawrence River has been classified as an area of concern. In its most recent data report, 52% of the sediment samples exceeded the draft sediment quality guideline for lead (St. Lawrence River Sediment and Biological Assessment: Data Report, 1990).

The sampling stations were in a highly industrialized section of the *St. Lawrence* where the severe effect level for lead was exceeded. For instance, in 1984 and 1985, 21% and 22% of the samples, respectively, exceeded the guideline of 31 µg/g.

Temporal and spatial information on the sediment quality in selected Great Lakes areas of concern is presented in Table D-2-3. Data were taken from individual survey reports on the respective area.

TABLE D-2-2 SEDIMENT BULK LEAD CONCENTRATIONS AT IN-PLACE POLLUTANT SITES: 1983-1987 (in order of decreasing sediment concentration)

LOCATION	LEAD CONCENTRATION (µg/g dry weight)	LOCATION	LEAD CONCENTRATION (µg/g dry weight)
Toronto Harbour	850*	Hamilton Harbour	570*
Ashbridges Bay	560*	Rice Lake, Midland Bay	
Toronto Harbour	460*	Toronto Harbour	510*
Ashbridges Bay (2)	420*	Toronto Main	430*
Hamilton Harbour	340*	Detroit River	360*
St. Mary's River	320*	Hamilton Harbour	330*
Toronto Harbour	290*	Hamilton Harbour, Toronto	300*
Toronto Harbour	270*	Toronto Harbour (2)	280*
		Midland Bay	260*

TABLE D-2-2 SEDIMENT BULK LEAD CONCENTRATIONS AT IN-PLACE POLLUTANTS SITES: 1983-1987 (in order of decreasing sediment concentration) (Continued)

LOCATION	CONCENTRATION (µg/g dry weight)	LOCATION	LEAD CONCENTRATION (µg/g dry weight)
Toronto Harbour	240	Detroit River, Toronto Harbour	220
Toronto Harbour, Humber Bay (2)	210	Hamilton Harbour	200
Humber Bay, Toronto Harbour	190	St. Mary's River	180
East Headland, Humber Bay (3)	170	Rice Lake, Toronto Harbour, Humber Bay (2)	160
St. Mary's River, Humber Bay	150	Ashbridges Bay, Humber Bay (3), Niagara River, Collingwood Harbour, East Headland (2), Grand River, Detroit River	140
Ashbridges Bay, St. Mary's River, Rice Lake, Penetang Harbour, Humber Bay (2)	130	Humber Bay (3), Ashbridges Bay, Midland Bay	120
St. Mary's River, Humber Bay (2), East Headland, Detroit River East	110	St. Clair River	108
Humber Bay	98	Humber Bay (3), Penetang Harbour	96
Humber Bay (2)	95	Midland Bay, Rice Lake, St. Mary's River	91
St. Mary's River	90	Collingwood Harbour	89
St. Mary's River, Toronto Main	82	Rice Lake, Niagara River, Frenchman's Bay	76
St. Mary's River	75	Midland Bay, Collingwood Harbour, Rice Lake, Niagara River	74
Grand River, St. Mary's River, Frenchman's Bay	72	Midland Bay, Penetang Harbour	71
Humber Bay (2)	70	Bluffers Park (2)	66
Humber Bay, Bluffers Park	65	Rice Lake	64
Midland Bay	63	Midland Bay, Rice Lake, Niagara River, Ashbridges Bay	62

TABLE D-2-2 SEDIMENT BULK LEAD CONCENTRATIONS AT IN-PLACE POLLUTANTS SITES: 1983-1987 (in order of decreasing sediment concentration) (Continued)

LOCATION	CONCENTRATION ($\mu\text{g/g}$ dry weight)	LOCATION	LEAD CONCENTRATION ($\mu\text{g/g}$ dry weight)
East Headland, Penetang Harbour, Humber Bay, Midland Bay	61	Humber Bay, Bluffers Park	60
Rice Lake	59	East Headland (2)	57
Humber Bay (2), Niagara River, Collingwood Harbour, Port Weller, Hamilton Harbour (outer), East Headland	56	Hamilton Harbour (outer), St. Clair River	55
St. Mary's River (2), Oakville Harbour, St. Lawrence River	54	East Headland (2), Frenchman's Bay, St. Mary's River, Humber Bay	53
Niagara River	50	Collingwood Harbour	49
Bluffers Park, Detroit River	48	Rice Lake, Midland Bay	47
St. Lawrence River, Rice Lake, Frenchman's Bay, St. Clair River	44	Collingwood Harbour, Oakville Harbour	42
Toronto Harbour	40	Canagagigue Creek, St. Lawrence River	38
Penetang Harbour	37	Humber Bay (2), East Headland (2)	36
St. Lawrence River	35	St. Lawrence River (4), Toronto Main, Bluffers Park, St. Clair River	33
Rice Lake, Oakville Harbour	32	Detroit River, Rice Lake, Oakville Harbour, Hamilton Harbour (outer)	31
Midland Bay, Ashbridges Bay	30	Toronto Main, Canagagigue Creek	29
St. Mary's River	28	Collingwood Harbour, Oakville Harbour, Bluffers Park, St. Mary's River, Hamilton Harbour (outer), Canagagigue Creek	27
Toronto Main, Niagara River, East Headland, Oakville Harbour, Detroit River	26	Humber Bay (2), Bluffers Park, St. Mary's River, Canagagigue Creek, Rice Lake	25

TABLE D-2-2 SEDIMENT BULK LEAD CONCENTRATIONS AT IN-PLACE POLLUTANTS SITES: 1983-1987 (in order of decreasing sediment concentration) (Continued)

LOCATION	CONCENTRATION (µg/g dry weight)	LOCATION	LEAD CONCENTRATION (µg/g dry weight)
St. Clair River	24.5	Hamilton Harbour (outer), Frenchman's Bay	23
Port Weller, St. Lawrence River	22	Kam River (3), St. Lawrence River, Ashbridges Bay	21
Toronto Main, Humber Bay (3), Collingwood Harbour (2), Ashbridges Bay, Canagagigue Creek	18	St. Clair River (4), Kam River, St. Mary's River	17
Niagara River, St. Clair River (2), Oakville Harbour, Grand River, Toronto Main (2), Rice Lake, Kam River, Collingwood Harbour	16	Toronto Harbour, Niagara River, St. Lawrence River, Kam River	15
Canagagigue Creek, Niagara River, Oakville Harbour	14	St. Clair River (2), Toronto Main	13
Bluffers Park, St. Clair River (2), Humber Bay, St. Lawrence River	11	Niagara River, Toronto Main, Toronto Harbour, St. Lawrence River, Penetang Harbour, Collingwood Harbour	10
Bluffers Park	9.6	Niagara River, Humber Bay (2)	9.1
St. Clair River (2)	8.5	St. Clair River	8.1
Collingwood Harbour	8	East Headland (2)	7.8
St. Clair River	7.5	St. Clair River	6.7
East Headland (2)	4.9	Bluffers Park	4.7
Port Weller	4.6	St. Mary's River	4.5
Toronto Main	4.4	St. Mary's River	4.1
Ashbridges Bay	3.4	East Headland (2)	3.3
Bluffers Park	3.2	Port Weller, East Headland (2)	3
Ashbridges Bay	2.3	Penetang Harbour	2
Bay of Quinte (8), East Headland, Niagara River	<2		

(#) number of stations * stations surpassing the severe effects level

Source: Draft VII, In-Place Pollutants Program of Ontario. (MOEE, 1991e)

TABLE D-2-3 SEDIMENT LEVELS OF LEAD IN SELECTED GREAT LAKES AREAS OF CONCERN

AREA OF CONCERN	LEAD LEVELS ($\mu\text{g/g}$)		REFERENCE
	MEAN (Maximum)	RANGE	
Niagara River	133		Remedial Action Plan for The Niagara River (Ontario) Area of Concern (Sept., 1990)
Bay of Quinte 1990	118 max (4 samples)		Bay of Quinte Remedial Action Plan: Stage 1 Environmental Setting & Problem Definition (July, 1990)
St. Lawrence 1990 1985 1984 1975 1979-1982 Upstream North Shore South Shore	59.7 25.62 max 130 max 190 10 118 18	8.4-520 2.3-270 5.5-12 16-1600 <3-53	Data Report: St. Lawrence River Sediment and Biological Assessment (WRB, 1990); St. Lawrence River Environmental Investigations; Vol 1 (Feb, 1988) Vol 3 (Feb, 1988) Vol 4 (Oct, 1990)
Port Hope 1984 Lake Ontario West Slip Ganaraska Confluence 1985 Ganaraska	 9 max 19 127 max 196 235 n/a 9 max 10 44.7	 21-69	Port Hope Harbour Remedial Action Plan: Stage 1 Environmental Conditions and Problem Definition (Jan, 1990)

TABLE D-2-3 SEDIMENT LEVELS OF LEAD IN SELECTED GREAT LAKES
AREAS OF CONCERN (Continued)

AREA OF CONCERN	LEAD LEVELS ($\mu\text{g/g}$)		REFERENCE
	MEAN (Maximum)	RANGE	
Hamilton Harbour 1975 1976 1977 1980 1986 *all exceeded the guideline	320 260 310 (May) 310 (Oct) 310 281		Remedial Action Plan for Hamilton Harbour: Environmental Conditions and Problem Definition (Mar, 1989)
Severn Sound 1980 - 1985		<2 - 260	Severn Sound Remedial Action Plan: Environmental Conditions and Problem Definitions (Feb, 1989)
Wheatley Harbour ¹ 1986 - 1987	35-109	10-49	Remedial Action Plan Wheatley Harbour: Stage 1 Environmental Conditions and Problem Definition (Apr, 1990)
Metro Toronto Humber Bay Toronto Harbour Toronto East Headland Eastern Toronto Waterfront	104 127 83 17		Metro Toronto Remedial Action Plan: Stage 1 Environmental Conditions and Problem Definition (May, 1989)

¹ Monitoring done by two different agencies - methods were not consistent

D-2.2.2 Levels of Lead in Ontario Drinking Water

The level of lead found in drinking water depends on:

- the amount of lead in the source water, and
- the amount of lead introduced by the distribution system.

Water entering the system may already contain lead from geologic deposits, aerial deposition or other contamination routes. If the distribution system contains lead pipes or solder, this lead may leach into the water. This section examines the lead concentrations found in Ontario drinking water, in ground and surface waters and in distribution systems.

D-2.2.2.1 Lead in Ground Water Supplies

Many municipal and most private drinking water supplies rely on ground water as their source. Lead concentrations would depend on the water chemistry and on the geochemical composition of the water-bearing strata.

Ground waters, at least in southern Ontario, are reasonably hard with a neutral to alkaline pH, factors which tend to make the water less corrosive with respect to lead. It has been suggested that acid rain may affect ground water quality where the overburden does not buffer recharge water (Meranger *et al.*, 1984). However, studies done by the Ontario Ministry of the Environment in the Muskoka, Haliburton and Sudbury areas, have shown that water quality has not been appreciably affected by acid rain (Sibul and Reynolds, 1982; Sibul and Vallery, 1982). Lead levels found in water sample from the Muskoka-Haliburton area were all less than 30 µg/L. In the Sudbury area, 42 ground water wells had background lead levels (flushed samples) ranging from less than 3 to 16 µg/L, with most values below 6 µg/L.

In south central Ontario, over 100 wells were tested to determine if ground waters were sensitive to acid rain. Water samples taken directly from 52 of the wells usually had lead concentrations less than 3 µg/L. Three wells had values above 6 µg/L, with the maximum reported value being 14 µg/L.

Lead levels were also relatively low in municipal drinking water systems that were ground water supplied. Twenty of the 75 municipal drinking water systems sampled in the Water Distribution Surveys (1981-1987) were ground water supplies. The majority had lead concentrations of less than 3 µg/L; three systems had raw values of 10 µg/L or less.

D-2.2.2.2 Lead in Surface Water Supplies

Most municipal water supplies in Ontario use surface waters as a source. Larger communities use a conventional treatment train that includes coagulation, flocculation, settling, filtration

and disinfection. Smaller communities, remote from sources of pollution, may only have minimal treatment with disinfection.

Since 1984, the Drinking Water Surveillance Program has monitored raw water quality at approximately 50 surface water treatment plants (WTP). The majority use water from the Great Lakes but some use water from inland lakes and rivers in both southern and northern Ontario. Almost without exception, lead levels are very low. Of the 603 results reported from 1985 to 1987, 559 had lead concentrations below 6 µg/L; 32 had concentrations between 6 and 11 µg/L; 11 had concentrations between 11 and 20 µg/L; and only one exceeded 20 µg/L. The levels have continued to decrease from 1988 through to 1991.

D-2.2.2.3 Lead in Tap Water

Municipal drinking water data on lead levels at consumer's taps are available from four recent Ontario studies: Distribution Systems Surveys; the Northern Ontario Blood Lead Study; the Drinking Water Surveillance Program; and the Composite Sampler Study (all unpublished MOEE data).

Lead concentrations in tap water will depend on the type of sample taken. The Ontario Drinking Water Objectives are guidelines for free-flowing tap water. Such running water or "flushed" samples invariably contain the lowest lead concentrations, because the water has had minimal contact with the house plumbing. The highest concentrations are found in "standing" samples, where water has been in contact with the plumbing for a longer period of time. The usual time period is 6 to 8 hours, or overnight. Sometimes a random sample may be taken during the day to estimate typical daytime concentrations.

The amount of lead consumed in drinking water is difficult to estimate. Different types of water samples are taken to estimate the lead concentrations. Furthermore use patterns of each consumer will vary. Typically, water usage is estimated to be at 400 L/person/day, with the actual water consumption estimated at only 1.5 L/person/day. An on-site integrated pump sampler has been used in the Montreal area to determine the total daily intake of lead. The intake of lead from drinking water was found to be 0.59 to 0.85 µg/day (Meranger *et al.*, 1984).

Distribution System Surveys (1981-1987)

The surveys were conducted on 77 municipal water systems throughout Ontario and are considered representative of the province. Twenty of the systems used ground water supplies, and the rest surface water. Samples were taken at each location over a restricted time period of a few days from both the raw and treated water at the plant. In addition, samples were taken from the taps of about five to seven consumers. Flushed water samples

were collected from each consumer, as well as random standing or overnight standing samples. Samples were usually taken from the kitchen tap.

The lowest lead concentrations were found in the flushed samples (Table D-2-4). The mean median concentration was 38 $\mu\text{g/L}$, with a range of 1 to 65 $\mu\text{g/L}$. Only one median value was above 8 $\mu\text{g/L}$. Most of the values were below the analytical detection limit (1 to 3 $\mu\text{g/L}$) but the detection limit was used to derive the mean.

A random standing sample was frequently taken prior to flushing. As expected, lead concentrations in these samples were usually higher than in the flushed samples. Of the 69 locations where random standing samples were taken, seven had median values above 50 $\mu\text{g/L}$ (range 1-423 $\mu\text{g/L}$).

The mean median concentration was 28.2 $\mu\text{g/L}$, with a range of 1 to 423 $\mu\text{g/L}$. The mean median value, however, is not representative of standing samples in the province, since many random standing samples were taken only where high lead levels were expected.

Overnight standing samples were taken at 32 locations. The mean median concentration was 9.1 $\mu\text{g/L}$, with a range of 1 to 49 $\mu\text{g/L}$.

A summary of the data on the 77 distribution systems surveys is presented in Table D-2-5 including residences with copper plumbing and lead services (pipes conveying potable water from the public water main to the inside of a building). Median flushed concentrations were below or just above the detection limit of 3 $\mu\text{g/L}$ in all cases. For the random and overnight standing samples, lead concentrations from residences with copper plumbing did not exceed general levels. In contrast, residences with lead services had lead concentrations one order of magnitude higher (10 to 17 $\mu\text{g/L}$).

TABLE D-2-4 LEAD CONCENTRATIONS (µg/L) IN TAP WATER FROM ONTARIO COMMUNITIES IN THE DISTRIBUTION SYSTEMS SURVEY

LOCATION	FLUSHED			RANDOM			OVERNIGHT		
	N	MED	RANGE	N	MED	RANGE	N	MED	RANGE
Aurora	5	3	3-4	2	4	4-4	0	-	-
Bancroft	5	3	3-3	0	-	-	0	-	-
Barry's Bay	7	2	2-2	7	2	2-10	7	9	2-18
Beeton	1	5	5	1	7	7	1	7	7
Belleville	5	3	3-4	2	39	10-68	0	-	-
Bracebridge	6	5	3-24	1	38	38	0	-	-
Brantford	10	3	3-12	6	7	3-12	0	-	-
Burk's Falls	4	3	3-4	2	423	26-820	0	-	-
Cache Bay	4	3	3-3	1	210	210	0	-	-
Caledonia	4	4	3-4	2	4	3-4	0	-	-
Callander	4	3	3-3	2	10	9-10	0	-	-
Campbellford	5	3	3-5	2	5	4-5	0	-	-
Campbellville	3	7	5-11	0	-	-	0	-	-
Capreol	7	2	2-32	7	2	2-31	7	20	2-76
Cayuga	0	-	-	1	30	30	0	-	-
Chapleau	3	5	3-9	0	-	-	0	-	-
Cobalt	6	3	3-5	3	59	4-110	0	-	-
Cookstown	3	3	3-4	3	3	3-7	3	3	3-7
Delhi	5	3	3-3	2	3	3-3	0	-	-
Deseronto	2	3	3-3	3	3	3-5	0	-	-
Dryden	7	2	2-6	7	2	2-2	7	2	2-2
Dunnville	3	3	3-6	2	15	12-18	0	-	-
Dymond Township	4	3	3-3	2	62	3-120	0	-	-
Elliot Lake	7	2	2-6	7	2	2-5	7	2	2-9
Elora	5	3	1-4	0	-	-	0	-	-
Erin	1	3	3	3	30	4-30	2	22	4-40

TABLE D-2-4 LEAD CONCENTRATIONS ($\mu\text{g/L}$) IN TAP WATER FROM ONTARIO COMMUNITIES
IN THE DISTRIBUTION SYSTEMS SURVEY (Continued)

LOCATION	FLUSHED			RANDOM			OVERNIGHT		
	N	MED	RANGE	N	MED	RANGE	N	MED	RANGE
Essex	2	5	5-5	2	5	4-5	2	7	7-7
Fenelon Falls	6	3	3-3	0	-	-	0	-	-
Gananoque	11	8	2-21	11	13	2-41	7	16	11-52
Goderich	8	3	1-8	8	11	1-30	8	9	1-47
Gravenhurst	5	3	3-4	0	-	-	0	-	-
Haileybury	6	3	3-3	3	11	3-17	0	-	-
Hamilton	5	4	3-8	2	17	12-22	0	-	-
Hastings	5	3	3-3	1	15	15	0	-	-
Hearst	8	1	1-21	8	4	1-45	8	1	1-48
Huntsville	5	3	3-3	2	4	3-4	0	-	-
Kenora	7	2	2-8	7	2	2-18	7	7	4-118
Kingsville	5	3	3-3	1	7	7	0	-	-
Kirkland Lake	8	2	1-5	7	10	5-70	7	21	3-68
Latchford	4	3	3-3	1	29	29	0	-	-
Leamington	6	7	1-16	3	5	4-32	0	-	-
Lindsay	5	3	3-22	1	100	100	0	-	-
Marmora	5	3	2-4	0	-	-	0	-	-
Mattawa	6	3	3-3	3	19	12-46	0	-	-
Milton	2	3	3-3	2	3	3-3	2	6	3-9
Mount Forest	4	3	1-3	2	4	3-4	0	-	-
Nanticoke	5	3	3-3	1	3	3	0	-	-
New Liskeard	6	3	3-7	3	8	3-10	0	-	-
Newmarket	2	3	3-3	3	3	3-3	3	3	3-3
Nobel	4	3	3-4	2	9	7-10	0	-	-
North Bay	10	3	3-13	7	37	7-120	0	-	-

TABLE D-2-4 LEAD CONCENTRATIONS ($\mu\text{g/L}$) IN TAP WATER FROM ONTARIO COMMUNITIES
IN THE DISTRIBUTION SYSTEMS SURVEY (Continued)

LOCATION	FLUSHED			RANDOM			OVERNIGHT		
	N	MED	RANGE	N	MED	RANGE	N	MED	RANGE
Ottawa	7	2	2-3	7	2	2-10	7	4	2-24
Owen Sound	8	1	1-4	8	1	1-5	7	1	1-9
Parry Sound	6	3	3-3	4	29	4-110	0	-	-
Pembroke	7	2	2-2	7	3	2-12	7	9	2-63
Peterborough	5	3	3-8	1	12	12	0	-	-
Pictou	2	3	3-3	2	3	3-3	3	3	3-3
Plattsville	2	65	64-66	3	12	5-45	3	49	35-65
Powassan	4	3	3-3	2	6	3-8	7	3	2-9
Rainy River	7	2	2-3	7	2	2-8	7	3	2-9
Sault Ste-Marie	7	2	2-3	6	2	2-3	7	4	2-152
Sioux Lookout	7	2	2-2	7	14	8-28	7	18	12-34
South River	6	3	3-6	2	340	310-370	0	-	-
Strathroy	7	2	2-26	7	2	2-2	7	2	2-2
Sturgeon Falls	6	3	3-3	4	48	9-88	0	-	-
St-Thomas	7	2	2-2	7	2	2-2	7	2	2-2
Sudbury	7	2	2-38	7	2	2-102	7	21	2-90
Temagami North	4	3	3-3	2	49	29-68	0	-	-
Temagami South	3	3	3-3	2	62	3-120	0	-	-
Thorne	4	3	3-5	3	34	10-51	0	-	-
Thunder Bay North	7	2	2-19	7	10	3-44	7	16	5-46
Thunder Bay South	7	2	2-20	7	7	3-22	7	17	4-37
Timmins	8	1	1-3	8	3	1-15	7	2	1-18

TABLE D-2-4 LEAD CONCENTRATIONS ($\mu\text{g/L}$) IN TAP WATER FROM ONTARIO COMMUNITIES IN THE DISTRIBUTION SYSTEMS SURVEY (Continued)

LOCATION	FLUSHED			RANDOM			OVERNIGHT		
	N	MED	RANGE	N	MED	RANGE	N	MED	RANGE
Trenton	6	3	3-3	0	-	-	0	-	-
Verner	4	3	3-33	2	20	3-37	0	-	-
Warton	7	2	2-2	7	2	2-2	7	2	2-2
Windsor	8	1	1-8	7	2	1-13	7	1	1-8

TABLE D-2-5 SUMMARY OF LEAD CONCENTRATIONS IN ONTARIO TAP WATER BY TYPE OF PLUMBING SYSTEM

	NUMBER OF SAMPLES	MEAN ($\mu\text{g/L}$)	STANDARD DEVIATION	MEDIAN ($\mu\text{g/L}$)	RANGE ($\mu\text{g/L}$)
<u>FLUSHED SAMPLE:</u>					
All data	408	4.1	6	< 3	1-66
Lead service connection	36	9.2	9.2	4	1-38
Copper plumbing	240	4	7.2	< 3	1-66
<u>RANDOM SAMPLE:</u>					
All data	264	19.5	62.4	4	1-820
Lead service connection	36	21.8	26.8	10	1-102
Copper plumbing	192	21	67.8	4	1-820
<u>OVERNIGHT SAMPLE:</u>					
Lead service connection	25	24.4	25.8	17	1-90
Copper plumbing	147	12.8	20.4	4	1-152

Source: Unpublished MOEE data.

Higher lead concentrations in the standing water samples were associated with low pH, low alkalinity, lead pipes or copper plumbing joined with lead-tin solder. Even with non-aggressive water, flushed samples from homes with lead service connections sometimes had increased lead levels.

Northern Ontario Blood Lead Study (1987-1988)

This study examined several environmental sources of lead, including drinking water, air, soil and dust in relation to the blood lead levels of children less than 6 years of age. About 150 homes were sampled in seven Northern Ontario communities. Sites with elevated lead levels in drinking water were deliberately selected.

The median lead concentrations in the flushed, random standing and overnight standing samples are reported in Table D-2-6. Additional data collected included the type of plumbing, general water chemistry, age of residence and when the plumbing work was done. Most concentrations, even in the standing samples, were low, with only two communities having median values above 10 µg/L. The maximum concentrations, however, were often elevated in all three types of samples. Elevated lead levels were associated with low alkalinity, and low pH, but not with the type or age of the plumbing.

**TABLE D-2-6 LEAD CONCENTRATIONS IN TAP WATER FROM SELECTED
NORTHERN ONTARIO COMMUNITIES**

LOCATION	FLUSHED SAMPLE			RANDOM STANDING SAMPLE			OVERNIGHT STANDING SAMPLE		
	N*	MEDIAN (µg/L)	RANGE (µg/L)	N*	MEDIAN (µg/L)	RANGE (µg/L)	N*	MEDIAN (µg/L)	RANGE (µg/L)
Hearst	32	3	3-43	30	3	3-49	29	3	3-64
Hymers	18	3	3-6	17	3	3-35	17	3	3-64
Moosonee	15	3	3-11	15	3	3-95	15	3	3-95
New Liskeard	15	3	1-9	15	3	3-13	14	3	3-9
North Bay	14	3	3-15	15	11	3-30	14	15	3-27
Sturgeon Falls	16	3	3-20	16	8	3-32	16	8	3-42
Thunder Bay	35	5	3-180	34	18	3-150	34	14	3-120

*number of houses sampled

Source: Unpublished MOEE data.

Drinking Water Surveillance Program: 1985-1991

Data for lead from the Drinking Water Surveillance Program (DWSP) are available as follows: 1985 (17 water treatment plants (WTP) sampled); 1986 (21 WTP); and 1987 (42 WTP).

All of the WTP sampled in 1985 and 1986 and most of those in 1987 were located in Southern Ontario and were supplied by surface water. Samples of the raw and treated water

were usually taken monthly at the WTP. Flushed and overnight standing samples were taken from the taps of two houses in the distribution system.

The means, median means and ranges for flushed and overnight samples from the houses sampled from 1985 to 1991 are included in Table D-2-7. Summary results are presented in Table D-2-8 below.

The lead concentrations in flushed samples have consistently decreased. It is not clear that the same decrease has occurred for overnight standing samples given the large ranges reported. Temporal data (1985-1991) for the mean flushed and overnight standing lead concentrations are given in Table D-2-9 for each sampling site.

TABLE D-2-7 LEAD CONCENTRATIONS ($\mu\text{g/L}$) IN TAP WATER FROM ONTARIO COMMUNITIES IN THE DRINKING WATER SURVEILLANCE PROGRAM

	N [*]	FLUSHED			OVERNIGHT		
		N ^{**}	MEAN MEDIAN	RANGE	N ^{**}	MEAN MEDIAN	RANGE
Belleville	2	19	4	3-20	19	6	3-22
Brantford	2	20	4	3-10	20	7	3-81
Burlington	2	19	3	3-5	20	4	3-54
Cornwall	2	18	3	3-8	18	5	3-10
Deseronto	1	6	3	3-8	6	3	3-6
Elgin - St-Thomas	2	19	3	3-5	18	3	3-7
Fort Erie	2	13	3	3-9	12	3	3-11
Grimsby	1	11	3	3-8	9	3	3-11
Hamilton	2	29	3	3-14	29	5	3-28
Kingston	2	35	3	3-5	37	3	3-22
London							
Lake Huron	2	27	3	3-9	21	18	3-400
Mississauga							
Lorne Park	2	31	3	3-6	31	3	1-18
Mississauga							
South Peel	2	137	3	1-7	137	3	1-58
Niagara Falls	2	66	3	3-11	63	3	3-8

TABLE D-2-7 LEAD CONCENTRATIONS ($\mu\text{g/L}$) IN TAP WATER FROM ONTARIO COMMUNITIES IN THE DRINKING WATER SURVEILLANCE PROGRAM
(Continued)

	N*	FLUSHED			OVERNIGHT		
		N**	MEAN/M EDIAN	RANGE	N**	MEAN/M EDIAN	RANGE
North Bay	2	16	4	3-15	18	16	3-460
Oshawa	2	20	3	3-12	20	4	3-7
Ottawa Britannia	2	32	3	3-4	31	6	3-21
Ottawa Lemieux	2	32	4	3-22	32	9	3-220
Peterborough	2	23	3	3-4	24	6	3-20
Port Dover	2	16	3	3-8	17	6	3-29
Sarnia Lam- bton	2	30	3	3-8	29	5	3-20
Sault Ste-Marie	2	16	3	3-12	16	8	3-14
Ste-Catharines	2	20	3	3-60	20	3	3-12
Sudbury Ramsay Lake	2	13	3	3-7	14	10	3-23
Sudbury Wanapitei	2	27	3	3-7	28	7	3-23
Toronto- Clark	2	10	3	3-4	11	3	3-5
Toronto Esterly	2	12	3	3-5	11	4	3-6
Toronto Harris	2	9	4	3-6	8	20	3-42
Union Leamington	2	20	3	3-8	20	4	3-37
Wallaceburg	2	35	3	3-9	36	4	3-1800
Windsor	2	4	3	3-18	40	4	3-11

* number of houses sampled

** number of samples taken

TABLE D-2-8 SUMMARY OF LEAD CONCENTRATIONS IN TAP WATER FROM THE DRINKING WATER SURVEILLANCE PROGRAM (1985-1991)

YEAR	LEAD CONCENTRATIONS (µg/L)			
	FLUSHED SAMPLE		OVERNIGHT STANDING SAMPLE	
	MEAN AVERAGE	RANGE OF MEAN	MEAN AVERAGE	RANGE OF MEAN
1985-87	3.0 to 4.0 ¹	(n.a.)	6.1 ¹	(3.0 -20.0) ²
1988	2.04	(0.13-37.0)	9.96	(0.79-63.2)
1989	1.02	(0.15-3.82)	8.63	(0.80-63.8)
1990	1.07	(0.12-15.5)	6.05	(0.20-45.3)
1991	0.47	(0.08-1.47)	5.84	(0.29-99.0)

¹ mean median ² range of mean median

TABLE D-2-9 LEAD CONCENTRATIONS (µg/L) IN THE TAP WATER FROM THE COMMUNITIES IN THE DRINKING WATER SURVEILLANCE PROGRAM (1985-1991)

LOCATION	FLUSHED SAMPLE					OVERNIGHT SAMPLE				
	1985- 1987*	1988	1989	1990	1991	1985- 1987*	1988	1989	1990	1991
Ajax WTP	N/A	0.190	0.316	0.241	0.155	N/A	1.100	0.963	0.878	0.515
Alvinston WTP	N/A	N/A	N/A	0.183	0.080	N/A	N/A	N/A	0.280	0.290
Amherstburg WSS	N/A	N/A	0.361	0.252	0.210	N/A	N/A	1.281	0.771	5.033
Atikokan WTP	N/A	1.550	0.975	1.443	0.557	N/A	7.233	4.067	16.600	6.807
Barrie (Centennial Park Well)	N/A	N/A	N/A	0.674	0.400	N/A	N/A	N/A	1.727	1.455
Barrie (Johnson St. Well)	N/A	N/A	N/A	0.496	0.518	N/A	N/A	N/A	5.775	2.375
Barrie (Tiffen Well)	N/A	N/A	N/A	0.535	0.282	N/A	N/A	N/A	0.877	1.242
Belle River WTP	N/A	N/A	N/A	0.448	0.302	N/A	N/A	N/A	1.704	1.210
Belleville WTP	4	0.853	0.326	0.166	0.144	6	2.951	1.905	1.384	1.382
Bracebridge WTP	N/A	N/A	N/A	N/A	0.985	N/A	N/A	N/A	N/A	N/A
Brantford WTP	4	1.982	3.818	1.916	1.367	7	18.381	48.458	20.604	99.000
Brockville WTP	N/A	N/A	N/A	0.239	0.222	N/A	N/A	N/A	0.932	0.535

TABLE D-2-9 LEAD CONCENTRATIONS IN THE TAP WATER FROM THE COMMUNITIES IN THE DRINKING WATER SURVEILLANCE PROGRAM (1985-1991)(Continued)

LOCATION	FLUSHED					OVERNIGHT				
	1985-1987*	1988	1989	1990	1991	1985-1987*	1988	1989	1990	1991
Burlington WTP	3	0.277	1.315	0.555	0.925	4	2.050	5.300	1.732	37.700
Casselman WTP	N/A	N/A	0.893	0.648	0.375	N/A	N/A	7.268	5.014	3.710
Cayuga WTP	N/A	0.521	0.350	N/A	N/A	N/A	3.043	5.300	N/A	N/A
Chatham WTP	N/A	0.430	1.013	0.781	0.395	N/A	5.085	5.966	4.395	7.200
Cornwall WTP	3	0.613	0.767	0.386	0.237	5	5.289	4.376	3.385	4.025
Delhi Spring Supply	N/A	N/A	N/A	0.482	0.324	N/A	N/A	N/A	3.578	2.340
Delhi WTP	N/A	N/A	N/A	0.293	0.146	N/A	N/A	N/A	0.841	1.076
Deseronto WTP	3	0.356	0.668	0.340	0.265	3	2.600	3.742	2.717	2.250
Dresden WTP	N/A	0.344	0.722	0.434	0.243	N/A	5.164	5.015	4.718	3.275
Dryden WTP	N/A	0.133	0.154	0.192	0.485	N/A	0.793	1.441	10.599	4.800
Dunnville WTP	N/A	N/A	N/A	0.185	N/A	N/A	N/A	N/A	0.200	N/A
Elmira Well Supply (North Aquifer)	N/A	N/A	N/A	0.385	1.890	N/A	N/A	N/A	8.373	6.350
Elmira Well Supply (South Aquifer)	N/A	0.717	0.955	N/A	N/A	N/A	6.933	11.100	N/A	N/A
Fort Erie (Rosehill WTP)	3	0.363	0.675	0.455	0.327	3	1.822	2.068	2.658	1.447
Fort Frances WTP	N/A	37.00	3.153	5.817	1.775	N/A	57.00	8.310	10.507	6.300
Gravenhurst WTP	N/A	N/A	N/A	0.970	0.557	N/A	N/A	N/A	7.875	6.333
Grimsby WTP	3	0.232	0.742	0.582	0.575	3	4.247	20.483	20.302	26.500
Guelph Well Supply	N/A	N/A	N/A	0.522	0.406	N/A	N/A	N/A	1.495	1.360
Haldimand/Norfolk WSS	N/A	N/A	0.788	0.467	0.225	N/A	N/A	4.931	2.929	2.300
Hamilton WSS	3	0.774	1.558	1.644	0.990	5	2.927	3.449	2.150	1.353
Harrow-Colchester WSS	N/A	N/A	N/A	0.183	0.360	N/A	N/A	N/A	0.741	1.142
Hawkesbury WTP	N/A	N/A	0.687	0.293	0.229	N/A	N/A	2.507	2.050	1.312
Kenora WTP	N/A	0.950	1.067	3.670	1.500	N/A	5.900	8.180	26.82	48.00
Kingston WTP	3	0.285	0.381	0.145	0.100	3	4.793	5.073	0.472	0.410
Kitchener Well Supply	N/A	0.246	0.548	0.195	0.150	N/A	5.806	8.482	2.040	1.200
Lindsay WTP	N/A	N/A	0.854	0.707	0.396	N/A	N/A	5.300	3.061	3.057

TABLE D-2-9 LEAD CONCENTRATIONS IN THE TAP WATER FROM THE COMMUNITIES IN THE DRINKING WATER SURVEILLANCE PROGRAM (1985-1991)(Continued)

LOCATION	FLUSHED					OVERNIGHT				
	1985-1987*	1988	1989	1990	1991	1985-1987*	1988	1989	1990	1991
London (Lake Huron WSS)	3	0.658	0.585	0.314	0.200	18	24.00	22.00	N/A	N/A
Manitouowadge Well Supply	N/A	N/A	N/A	N/A	0.333	N/A	N/A	N/A	N/A	2.400
Metro Toronto (Easterly WSS)	3	0.286	1.053	0.355	0.313	4	1.839	3.560	3.753	2.850
Metro Toronto (Harris WTP)	4	0.820	0.630	N/A	N/A	20	15.525	18.00	N/A	N/A
Metro Toronto (Clark WTP)	3	0.194	0.441	4.190	0.495	3	1.029	1.948	11.00	3.250
Milton Well Supply	N/A	N/A	N/A	1.286	0.733	N/A	N/A	N/A	2.863	1.525
Napanee WTP	N/A	N/A	N/A	N/A	0.220	N/A	N/A	N/A	N/A	0.790
Niagara Falls WTP	3	0.156	0.390	0.267	0.225	3	2.160	7.027	5.845	7.015
Nipigon WTP	N/A	N/A	N/A	N/A	0.108	N/A	N/A	N/A	N/A	1.613
North Bay WTP	4	3.600	2.879	1.916	0.650	16	25.25	27.39	23.065	8.767
Oakville WTP	N/A	N/A	N/A	0.148	0.108	N/A	N/A	N/A	4.742	2.875
Odessa WTP	N/A	1.740	1.350	1.004	0.290	N/A	8.878	5.100	2.5822	0.610
Oshawa WSS	3	0.315	0.753	0.317	0.307	4	2.675	3.439	2.308	1.850
Ottawa WSS (Britannia)	3	0.706	0.662	0.752	0.427	6	15.665	9.530	4.634	7.500
Ottawa WSS (Lemieux Island)	4	1.332	1.517	0.856	0.562	9	7.235	6.409	5.700	3.675
Owen Sound (Neath WTP)	N/A	N/A	N/A	0.451	0.308	N/A	N/A	N/A	2.742	1.970
Owen Sound Spring Supply	N/A	N/A	N/A	0.565	0.518	N/A	N/A	N/A	0.639	0.432
Peterborough WSS	3	1.031	1.254	1.405	0.658	6	8.019	9.330	6.287	7.875
Plantagenet WTP	N/A	N/A	N/A	0.292	0.120	N/A	N/A	N/A	0.878	0.668
Port Colborne WTP	N/A	N/A	N/A	0.530	0.226	N/A	N/A	N/A	2.187	2.340
Port Dover WSS	3	0.833	0.415	0.204	N/A	6	7.896	1.385	1.954	N/A
Port Hope WTP	N/A	N/A	N/A	N/A	0.648	N/A	N/A	N/A	N/A	4.282
Port Stanley WTP	N/A	N/A	N/A	0.398	0.210	N/A	N/A	N/A	1.118	0.910
Prescott WTP	N/A	N/A	N/A	0.589	0.313	N/A	N/A	N/A	5.771	6.625
Renfrew WTP	N/A	N/A	0.722	15.480	0.337	N/A	N/A	13.015	4.356	5.146

TABLE D-2-9 LEAD CONCENTRATIONS IN THE TAP WATER FROM THE COMMUNITIES IN THE DRINKING WATER SURVEILLANCE PROGRAM (1985-1991)(Continued)

LOCATION	FLUSHED					OVERNIGHT				
	1985-1987*	1988	1989	1990	1991	1985-1987*	1988	1989	1990	1991
Rockland WTP	N/A	N/A	N/A	0.452	0.275	N/A	N/A	N/A	2.747	1.355
Samia WTP	3	0.329	0.704	3.413	0.440	5	5.589	8.078	9.033	6.000
Sault Ste Marie WTP	3	1.735	2.043	2.246	1.465	8	10.900	25.295	10.837	13.025
Simcoe Spring Supply (First Ave)	N/A	N/A	N/A	0.371	0.222	N/A	N/A	N/A	0.455	0.698
Simcoe Spring Supply (North West One)	N/A	N/A	N/A	1.471	0.940	N/A	N/A	N/A	11.625	9.740
South Peel WSS (Lakeview)	3	0.823	0.558	0.429	0.428	3	2.553	2.256	1.976	1.958
South Peel WSS (Lorne Park)	3	0.211	0.495	0.360	0.135	3	2.450	0.799	1.093	0.795
St. Catharines	3	0.191	1.239	0.124	0.095	3	0.960	5.255	0.934	1.467
St. Thomas (Elgin)	3	0.531	0.509	0.558	0.265	3	2.072	2.303	3.053	1.335
Stoney Point	N/A	N/A	N/A	0.919	0.235	N/A	N/A	N/A	1.012	3.075
Sudbury WTP	3	1.583	N/A	N/A	N/A	10	18.746	N/A	N/A	N/A
Sudbury WSS	3	0.346	N/A	N/A	N/A	7	2.995	N/A	N/A	N/A
Tecumseh WTP	N/A	N/A	N/A	0.498	0.293	N/A	N/A	N/A	4.502	2.267
Thamesville Well Supply	N/A	1.123	1.409	4.167	1.350	N/A	7.745	63.816	45.333	3.550
Thunder Bay (Bare Point WTP)	N/A	0.807	1.190	1.452	0.967	N/A	2.400	2.755	10.741	9.900
Thunder Bay (Loch Lomond WTP)	N/A	5.946	0.665	0.341	0.210	N/A	63.215	3.447	10.463	1.310
Tilbury WTP	N/A	N/A	N/A	0.787	0.225	N/A	N/A	N/A	14.170	0.437
Trenton WTP	N/A	N/A	3.535	1.381	0.285	N/A	N/A	8.500	26.080	3.200
Trenton Spring Supply	N/A	N/A	0.849	0.458	0.335	N/A	N/A	2.172	1.253	0.820
Union WSS	3	0.367	0.493	0.392	0.240	4	3.905	2.920	3.111	1.553
Wallaceburg WTP	3	0.615	0.255	0.196	0.177	4	3.999	2.647	2.076	3.450
Welland WSS	N/A	N/A	2.119	2.225	1.450	N/A	N/A	11.482	16.667	15.500
Windsor WSS	3	17.534	1.180	1.077	0.642	4	57.296	3.825	4.142	4.525

* mean median; all other values arithmetic mean

Composite Samples from Consumers (1988)

This study sampled the tap water of six consumers in each of seven municipalities. Municipalities were selected to represent major population areas and communities where high lead levels had been found in previous surveys. The houses chosen were expected to have higher lead levels due to new plumbing or a lead service connection. Where possible, the houses coincided with those sampled in the Northern Ontario Blood Lead Study. At each house, flushed and overnight standing samples were taken from the kitchen tap. A composite sampler collected daily samples of a portion of the water used for consumption over a one week period.

Lead levels in flushed and standing samples were similar to those found in the other three surveys, with the flushed sample containing lower concentrations than the standing sample. The southern Ontario locations had considerably lower values than those in northern Ontario. Grab samples (either flushed or overnight standing) were variable and did not provide consistent minimum and maximum values.

Lead concentrations from the composite samples were usually much more consistent than the grab samples (flushed and overnight standing samples) even though sampling techniques were very similar. Lead concentrations in the composite samples were often between the flushed and overnight concentrations in magnitude. However, the flushed and standing values varied considerably due to differences in service lines and plumbing. The results from composite samples were also affected by these factors and by the water-use patterns of the residents but provided a much better estimate of the lead consumed. A comparison of the lead concentrations found by the two different studies at the same houses is given in Table D-2-10.

The data from the seven municipalities where the composite samples were taken are summarized in Table D-2-11. The grab samples (flushed and overnight standing) are based on an average of six values, one set from each house, while the composite average value is an average of the six weekly composite results. The latter weekly values are a mean of seven daily results.

The average composite results vary from 1.8 to 14.5 µg/L. The mean concentration from the seven locations is 6.9 µg/L. If the 42 houses sampled are grouped together, the average lead concentration over one week ranged from 1.1 to 30 µg/L, with a median of 4.8 µg/L.

TABLE D-2-10 COMPARISON OF LEAD CONCENTRATIONS IN TAP WATER AT THE SAME HOUSES PARTICIPATING IN THE NORTHERN ONTARIO BLOOD LEAD STUDY (NOBLS) AND THE COMPOSITE SAMPLER STUDY (CSS)

LOCATION	FLUSHED SAMPLE (µg/L)		OVERNIGHT SAMPLE (µg/L)		COMPOSITE (CSS) SAMPLE (µg/L)	
	NOBLS	CSS	NOBLS	CSS	AVERAGE*	RANGE**
Cache Bay						
House 1	3	1	3	10	4	3-6
House 2	3	5	30	8	8	2-15
North Bay						
House 1	3	2	27	10	5	1-12
House 2	15	2	16	44	5	4-7
House 3	3	5	18	2	6	1-10
House 4	3	8	14	46	7	2-15
Thunder Bay						
House 1	38	5	39	12	12	9-15
House 2	180	7	120	23	30	21-37
House 3	21	25	38	39	20	12-25
House 4	95	18	39	25	10	7-12

* Average of composite samples over 1 week

** Range of composite samples

Source: Unpublished data from MOEE.

TABLE D-2-11 LEAD CONCENTRATIONS IN TAP WATER FROM SELECTED ONTARIO COMMUNITIES

LOCATION	MEAN (µg/L)	MEDIAN (µg/L)	RANGE (µg/L)
<u>FLUSHED SAMPLE</u>			
Cache Bay	3.5	3.6	1.1-7.0
Etobicoke	1.0	1.0	1.0
North Bay	10.9	3.6	1.0-47
Peterborough	1.0	1.0	1.0
Scarborough	1.0	1.0	1.0-2.0
Sudbury	3.3	1.7	1.0-10.4
Thunder Bay	12.2	12.2	1.0-24.5
<u>OVERNIGHT SAMPLE</u>			
Cache Bay	20.2	17.3	7.6-41.5
Etobicoke	1.3	1.0	1.0-2.6
North Bay	26.4	18.9	2.0-69
Peterborough	3.8	5.0	1.0-6.0
Scarborough	1.0	1.0	1.0-1.2
Sudbury	7.5	3.0	1.5-19.1
Thunder Bay	22.0	23.7	2.3-39.2
<u>WEEKLY COMPOSITE SAMPLE</u>			
Cache Bay	6.2	6.2	3.57-9.37
Etobicoke	1.8	1.8	1.1-2.33
North Bay	7.1	5.4	3.5-16.14
Peterborough	3.6	2.8	2.16-7.97
Scarborough	4.1	4.1	2.27-5.11
Sudbury	9.7	5.7	1.06-27.9
Thunder Bay	14.5	12.4	1.96-30.13

Source: Unpublished MOEE data.

D-2.3 Effect of Lead on Aquatic Biota

Aquatic organisms, specifically fish, can be used as indicators of a contaminant's burden to the aquatic environment because of bioaccumulation. Toxic chemicals may be present in concentrations below the minimum analytical detection limit, but levels in aquatic life can be used as an indirect assessment of water quality. Sport fish represent the greatest source of human exposure through aquatic biota, but levels of lead in other aquatic organisms will also be discussed, as indicative of the general health of the aquatic environment.

Three programs are in place to examine concentrations of toxic chemicals in fish. The Open-Lake Fish Contaminants Program, sponsored by the Canadian Department of Fisheries and Oceans and the U.S. Fish and Wildlife Service, commenced in 1977. Annual measurements are made of several contaminants in forage fish (rainbow smelt, bloater chub) and top predator fish (trout, walleye). Forage fish feed mainly on plankton and top predator fish on other fish.

Other fish species such as splake, sculpin, coho salmon, and carp have been examined when dealing with specific problems. Whole fish homogenates are used to determine contaminant levels because fish accumulate contaminants in non-edible tissues. Fish size expressed as total length (U.S. program) or fish age (Canadian program) can be used as the dependent variable for measuring changes in contaminant levels over time (DeVault *et al.*, 1986; Hesselberg *et al.*, 1988).

The Nearshore Juvenile Fish Contaminants Surveillance Program was initiated by the Ontario Ministry of the Environment in 1975. This involved the collection and analysis of spottail shiners from nearshore and connecting channel locations, especially in the Great Lakes areas of concern. Spottail shiners are plentiful in the Great Lakes, allowing for inter- and intra-lake comparisons and for monitoring the effectiveness of remedial actions. The program also measures whole fish levels and can thus be used to assess temporal and spatial trends.

The Sport Fish Testing Program was developed and implemented by the Ontario Ministry of the Environment. The program analyzes fish samples for various toxic contaminants and provide data for the development of fish consumption guidelines (MOEE, 1981b). It differs from the other two in that samples of skinless dorsal muscle tissue are analyzed instead of whole fish. The fish consumption guidelines are based on this edible tissue. Ontario has set an interim lead guideline for fish of 1 µg/g (ppm) in the edible portion (muscle).

Other short term studies have been done on contaminants present in Great Lakes fish. This information will be included in the following section where available.

D-2.3.1 Levels of Lead in Sport Fish

In *Lake Ontario*, higher lead levels are found in the forage fish than top predator fish (Table D-2-12). In 1978, the highest lead concentration (0.4 ppm wet weight) was measured in whole yellow perch at the Toronto location (Hodson *et al.*, 1984).

Total whole body lead levels measured in fish from various locations in *Lake Erie* ranged from 0.10 to 0.20 ppm. Values did not differ much between species from different trophic levels (Hodson *et al.*, 1984). A temporal analysis of rainbow smelt showed that the only exceedance of the detection limit of 0.01 ppm occurred in 1980. The mean concentration of lead in whole rainbow smelt in 1980 was 0.21 ppm.

TABLE D-2-12 TOTAL WHOLE BODY LEAD CONCENTRATIONS IN LAKE ONTARIO FISH, 1978

LOCATION	PREDATORY FISH	FORAGE FISH	
	TROUT/SALMON (ppm)	YELLOW PERCH (ppm)	RAINBOW SMELT (ppm)
Eastern Basin	<0.1	0.19	0.12
Point Travers	<0.1	-	0.12
Cabours	<0.1	-	0.22
Port Hope	<0.1	-	-
Port Credit	<0.1	-	0.16
Credit River	<0.1	-	-
Niagara	-	-	0.09
Toronto	-	0.40	-

Source: Hodson *et al.*, 1984

The lead burden in fish from *Lake Huron* is less than 0.1 ppm. Mean concentrations of whole body lead in offshore fish were 0.08 ppm in bloater chub and 0.05 ppm in burbot (Environment Canada, 1991).

Lake Superior total lead concentrations of whole fish were determined in 1976 for bloater chub, burbot and trout. The mean concentrations (wet weight) are 0.06, 0.04 and 0.04 ppm, respectively (Hodson *et al.*, 1984).

Lead levels in sport fish from the *St. Lawrence River* have been monitored by the MOEE for several years, near the DuPont Canada Inc. alkyllead production plant in Maitland. As of 1985, Blue Church Bay, a sampling location downstream from the Maitland plant, was the only location out of the 65 tested with lead levels in the edible portion of fish above the 1

µg/g Ontario guideline. The concentrations of lead in skinless, boneless dorsal fillets of fish collected at Blue Church Bay from 1983 to 1986 are given in Table D-2-13.

Lead levels in the vicinity of Maitland have been decreasing since 1981 (Johnson and Cox, 1988; Wong *et al.*, 1988). After the plant ceased production in 1985, the concentrations of lead in fish dropped below the 1 µg/g guideline. A temporal analysis of alkyllead was carried out in Maitland on carp, white sucker and brown bullhead (Wong *et al.*, 1988). The concentrations of alkyllead in whole fish samples are contained in Table D-2-14. Levels have declined steadily in all three species. The high mean values for carp in 1986 were due to high concentrations (9 to 10 ppm) in two fish; the remaining 18 had concentrations below 0.1 ppm. The maximum concentrations for the three species were 4.207 ppm, 3.725 ppm and 1.135 ppm, respectively, but these values all occurred before the plant closure in 1985.

TABLE D-2-13 CONCENTRATION OF LEAD IN THE EDIBLE PORTION* OF SPORT FISH IN THE ST. LAWRENCE RIVER AT BLUE CHURCH BAY

YEAR	SPECIES	NUMBER OF FISH	CONCENTRATION OF LEAD* (ppm wet weight)	LENGTH OF FISH* (cm)
1983	Northern Pike	20	1.22 (0.53-2.72)	58.2 (43.0-69.5)
	White Sucker	20	2.64 (0.27-5.86)	43.7 (37.7-47.7)
	Yellow Perch	17	6.18 (0.45-15.8)	20.1 (14.2-27.7)
	Carp	--	not measured	---
1984	Northern Pike	20	0.77 (<0.59-2.23)	61.2 (39.3-78.2)
	White Sucker	20	1.27 (<0.60-4.42)	44.7 (35.4-49.6)
	Yellow Perch	29	0.95 (<0.61-2.39)	25.5 (23.3-29.3)
	Carp	20	5.62 (<0.60-78.0)	77.1 (67.4-87.9)
1985	Northern Pike	10	0.58 (0.10-1.08)	57.3 (47.9-80.9)
	White Sucker	5	0.62 (0.10-1.08)	46.4 (44.7-49.2)
	Yellow Perch	10	1.02 (0.05-3.19)	23.9 (22.4-27.0)
	Carp	--	not measured	---
1986	Northern Pike	19	0.130 (.004-.590)	62.2 (40.2-81.8)
	White Sucker	19	0.198 (.039-.541)	47.4 (35.1-59.2)
	Yellow Perch	20	0.102 (.016-.313)	20.6 (16.5-26.1)
	Carp	20	0.879 (.016-9.82)	72.7 (63.7-81.8)

* skinless, boneless dorsal fillet

mean; range is in brackets

Data from Johnson and Cox, 1988

TABLE D-2-14 CONCENTRATION OF ALKYL LEAD IN WHOLE FISH,
MAITLAND, 1981-1987

YEAR	CARP (ppm)	WHITE SUCKER (ppm)	BROWN BULLHEAD (ppm)
1981	4.207	0.218	-
1982	1.976	1.747	1.135
1983	0.804	3.725	-
1984	-	0.411	0.294
1986	0.988	0.230	0.054
1987	0.120	0.050	0.149

Source: Wong *et al.*, 1988

Monitoring has also been done in the *St. Clair River*, near the Ethyl Canada Inc. plant in Corunna. The plant is still operating, but no unsafe lead levels have been found in fish from the river. In 1983, lead concentrations in fish were elevated near the alkyllead plant. Although the geometric means were below the 1 µg/g guideline, the ranges of the values in some of the fish were above this level (Wong *et al.*, 1988). Average alkyl lead concentrations in whole body carp and white suckers were 0.283 ppm and 0.138 ppm, respectively (Wong *et al.*, 1988). These lead concentrations declined in 1984, and again in 1987, with the means and ranges below the guideline.

Whole fish data cannot be used to evaluate the edibility of fish. Sites of lead accumulation in fish seem to depend on the species of lead. For most species, concentrations of tri- and dialkyllead in muscle are equivalent to those in the whole fish, although in white suckers and carp, levels are higher in muscle. Whole fish data, however, include the gut contents, which can be extremely variable in lead concentration. There is no relation between the distribution of lead in the gut contents and in muscle so that no conclusions regarding the lead content in fish muscle can be drawn from whole fish data.

D-2.3.2 Levels of Lead in Other Aquatic Organisms

Contaminant monitoring data for invertebrates are available for Lakes Ontario, Erie, Huron, and Superior. The Ontario Ministry of the Environment initiated an in-place pollutants program in 1983. As part of this program, benthic organisms were analyzed for contaminants, including lead. Data are available for 1983 for several nearshore and connecting channel locations, including the Toronto Waterfront, Hamilton Harbour, St. Mary's River, St. Clair River, Niagara River and St. Lawrence River (Persuad *et al.*, 1987).

Contaminant burdens in the Great Lakes were measured in surface plankton from 1977 to 1983. Surface plankton are mostly comprised of microscopic plants and zooplankton, such as the freshwater shrimp *Mysis relicta* and the amphipod *Pontoporeia* sp. *Mysis relicta* feeds off the water column and *Pontoporeia* sp. is a benthic organism feeding on the sediments and sedimenting material (Pennak, 1978).

Measured *Lake Ontario* lead concentrations in plankton fluctuated from a level of about 7 ppm (dry weight) in 1977, to below 6 ppm in 1978, and peaking in 1979 at a level of approximately 15 ppm. The estimated trend from 1979 to 1982 is downward to 4 ppm. The same temporal trend was observed for *Pontoporeia*. Levels were recorded at 2.5, 3.5, and 3 ppm for 1978, 1979, and 1980, respectively. From 1980 to 1982, the trend was downward to just below 2 ppm (Environment Canada, 1991). On a spatial basis, mean concentrations of lead were higher in *Pontoporeia* collected from the western basin of Lake Ontario compared to those from the eastern basin. The measured levels were 4.5 and 2.9 ppm, respectively (Whittle and Fitzsimons, 1983).

In *Lake Huron*, samples of plankton, *Mysis*, and *Pontoporeia* were collected from up to five locations during 1980 and 1983. The 1983 data demonstrate that lead concentrations were significantly higher in the North Channel than at the Goderich and South Baymouth sites. This may be a function of differing geology as well as different industries (mining and smelting). The measured 1983 levels were 3.8, less than 1.0, and 2.2 ppm for plankton, *Mysis*, and *Pontoporeia*, respectively (Environment Canada, 1991).

In 1983, *Lake Superior* sampling was carried out in Thunder Bay and Whitefish Bay. The level of lead in plankton was measured at 20.3 ppm and 5.2 ppm, respectively. Sampling for *Mysis* and *Pontoporeia* was carried out at Whitefish Bay only, resulting in lead levels of less than 0.5 ppm and 2.4 ppm, respectively (Environment Canada, 1991).

Concentrations of lead in amphipods from the *St. Lawrence River* near Maitland in 1984 were similar to those in the fish, ranging from 0.42 µg/g at an upstream control site to 5.21 µg/g at Blue Church Bay (Hayton, 1988).

APPENDIX D-3

LEAD IN THE TERRESTRIAL ENVIRONMENT

APPENDIX D-3 LEAD IN THE TERRESTRIAL ENVIRONMENT

D-3.1 Fate of Lead in the Terrestrial Environment

Lead is naturally present in the earth's crust and the natural presence of lead in soil is strongly related to the composition of the bedrock. Therefore, natural background soil lead levels are expected. It has been stated that Canadian soil lead background is probably less than 50 ppm (de Treville, 1964). This falls in the generally accepted typical background soil lead range of 2-200 ppm (Swaine, 1955 cited by Adriano, 1986; Zimdahl and Skogerboe, 1977).

Lead occurs in four valence states, Pb(0), Pb(I), Pb(II) and Pb(IV), and reacts to form compounds complexes and alloys rather than remain in its elemental form (Stokinger, 1981). Pb(II), the predominant and most stable valency, generally binds to ligands which provide multiple binding sites allowing for the formation of heterocyclic chelate rings. The geochemical characteristics of Pb^{2+} resembles the divalent alkaline earth group hence it has the ability to replace or displace Ba, Sr, and Ca in minerals and sorption sites (Kabata-Pendias and Pendias, 1984). Pb(IV) reacts readily with organic molecules providing a single or monodentate binding site. Salts found in mineral ores are the most common with galena (PbS) being the predominant compound.

The major source of soil lead is atmospheric deposition from predominantly anthropogenic sources such as smelting operations and the historical use of lead in automobiles. Occurring less frequently, elevated soil lead levels may be a result of illegal dumping of lead containing materials (ie. lead-acid batteries). Lead deposited to soil that originate from smelters occur mainly in the mineral forms (PbS, PbO, $PbSO_4$, $PbO \cdot PbSO_4$). Automobile exhaust emits lead in the form of halide salts (PbBr, PbBrCl). These compounds are often converted to lead sulphates in the atmosphere or in soil due to the instability of the halide compounds (Zimdahl and Skogerboe, 1977; Kabata-Pendias and Pendias, 1984).

Accumulation of lead in soil is generally greater at the surface which could be due to either atmospheric deposition or the tendency of surface layers to have greater amounts of organic matter that will immobilize lead (Adriano, 1986). Lead has been shown to migrate downward to deeper soil layers. This leaching tendency can be attributed to different processes: 1) soluble chelate complexes 2) transfer of soil particles by earthworms and other faunal organisms 3) translocation in plant roots. Horizontal movement can be attributed to tillage and wind (Adriano, 1986).

Most lead is retained strongly in soil, but more soluble forms may migrate into surface or ground water (ATSDR, 1988). Generally lead remains insoluble in soil likely due to the predominance of lead sulphide (PbS) which is fairly insoluble (Adriano, 1986). The lead salts vary in their solubility; lead chlorate, nitrate and acetate are soluble; lead chloride is slightly soluble; lead carbonate, chromate, phosphate and sulphate are insoluble.

Depending on the lead species and the soil parameters, lead can remain immobilized or move through/within the soil medium. Many factors contribute to the fate and transport of lead in soil some of which include: lead species introduced to medium; pH (optimal outside pH range 6-8); organic matter content; ion-exchange characteristics; presence of inorganic colloids and iron oxides; presence of microorganisms; uptake by plants; and precipitation.

There is an evident pH effect that occurs with lead in soil. Generally, lead solubility and extractability decreases with increasing soil pH. It has been shown that basic soils of arid and semi-arid areas will tend to accumulate lead (Adriano, 1986). The order of fractionation in arid soil is carbonate >> sulphide > organic matter. High soil pH may precipitate lead as hydroxide, phosphate or carbonate. These precipitates are insoluble and would therefore remain immobilized.

Fixation of lead in soil by precipitation can be significant at high pH's especially in calcareous (limestone) soils where carbonate (PbCO_3) may assume complexation importance. This agrees with the finding that lead carbonate constitutes a significant fraction of lead in basic arid and semi-arid soils (Adriano, 1986).

Although there are many fixation processes the primary and most significant process is that of adsorption by essentially insoluble organic matter. The composition of organic matter and its properties depends upon climate conditions, soil types and agricultural practices. Humic substances are the products of biological decay and these substances react readily with metal ions and of particular importance is its ability to form complexes with cationic species like Pb^{2+} . Some of the interactions/processes of metals and organic matter include ion-exchange, surface sorption, and chelation.

Sorption of lead by hydrous oxides may be through their collection of or by organic matter. Organic matter serves as a fixation medium for Fe, Mn and Pb oxides by binding readily to these metals and Fe and Mn oxides particles serve as accumulators for both lead and organic matter. The surface of these oxides can be hydroxylated to create a charge which then binds lead. This explains the high sorption capacity Mn and Fe have for Pb.

Solubilization of lead has been attributed to the formation of soluble chelate complexes with organic matter hence possibly attributing to the downward movement of lead. The solubility of the lead-complex formed was found to be inversely related to pH and the concentration of salt (Adriano, 1986).

The fixation capacity of organic matter has been illustrated in plant studies where amending the soil with organic matter decreased the lead uptake to plants (Zimdahl and Foster, 1976).

The clay content of soil has been shown to impact on the sorption of lead by soil particles. Generally, soil having a greater clay contents have a greater cation-exchange-capacity (CEC) hence a greater binding affinity for cations like Pb^{2+} . With an increase in CEC follows and increase in soil sorption of lead and a decrease in lead uptake to plants. In exchange

adsorption studies Pb^{2+} adsorption was favoured over Ca^{2+} in three different clay minerals, montmorillonite, illite and kaolinite (Bittell and Miller, 1974 as cited in Adriano, 1986).

The oxidation of sulphides (PbS) by weathering will promote the formation of carbonate and the incorporation into clay minerals, Fe and Mn oxides and in organic matter (Kabata-Pendias and Pendias, 1984).

Microbiological fixation of lead is also possible. It has been shown that increases in lead levels in soil are likely to limit enzymatic activity of microbiota hence increasing the accumulation of incompletely decomposed soil organic matter. Under certain conditions lead has been shown that it can be transformed by microorganisms as is the case in the formation of volatile tetramethyl lead by sediment microorganisms (Adriano, 1986).

Another possible movement within the terrestrial environment is uptake of lead by plants. Liming of soils decreases the availability of lead to plants (Zimdahl and Skogerboe, 1977) by decreasing its solubility in soil (Kabata-Pendias and Pendias, 1984) suggesting that the bioavailability of lead decreases in soil with higher pH's. This is evidenced by the tendency for lead to be taken up into plant roots when grown in slightly acidic soil. Other studies report a tendency for soil microflora to accumulate lead at a very high rate that was proportional to the metal content of soils (Niyazova and Letunova as cited in Kabata-Pendias and Pendias, 1984). Unless the plants are removed, lead will return to the soil when the plant decays (ATSDR, 1988).

There is evidence that atmospheric lead enters soil as lead sulphate ($PbSO_4$) or is converted rapidly to the sulphate at the air-soil surface (ATSDR, 1988). Lead sulphate is relatively insoluble so that lead would not likely leach if not transformed. The chemical composition of wet precipitation may affect lead by possibly converting it to a more soluble form. Acid rain, for example, tends to remove lead from the atmosphere and to solubilize it before it is deposited in soil or water. Acid rain could also solubilize the lead already present in soil, allowing it to leach into ground water.

D-3.2 Levels of Lead in the Terrestrial Environment

The Phytotoxicology Section, Air Resources Branch, Ontario Ministry of the Environment is in the process of developing Ontario Typical Ranges (OTR's) for certain chemical parameters in soil, vegetation and snow. The OTR's represent the expected range of concentrations for given contaminants in surface soil, snow or specific groups of plants. Samples are taken from areas in Ontario that are removed from known point sources of emission. The areas selected for sampling represent different land use categories based on the level of human activity (MOEE draft, 1991f).

OTR values are to be used to interpret analytical data and to evaluate source-related impacts. In other words, they will be used to determine whether the level of a contaminant in surface

soil, snow or plants is significantly higher than expected for the normal level of human activity in the area sampled. An action level, OTR_{98} , has been defined; it represents 98% of the data in the OTR distribution. Recently the lead OTR 's for rural and urban were determined to be 44.9 $\mu\text{g/g}$ and 98 $\mu\text{g/g}$ respectively (MOEE, unpublished data 1992f).

This section summarizes the available data on concentrations of lead in Ontario soils, and provides descriptions of both the soil and the adjacent environment from which the samples were obtained. Many factors, both natural and anthropogenic, may affect soil lead concentrations. Quantification of soil lead concentrations requires a clear definition of the conditions under which the various data were acquired, including the adjacent environment.

Lead concentrations in undisturbed soil are highly dependent on the depth at which the sample was taken. This is especially true in areas where lead accumulates from aerial sources. Thus, concentrations in the uppermost layer of soil will be much higher than in aggregate samples from a lower depth.

The following environments are sufficiently distinct to warrant separate discussion of soil lead conditions: rural remote, rural agricultural, urban residential, urban roadside, and urban industrial. Where data are not available, soil lead concentrations can only be inferred.

D-3.2.1 Lead in Soil - Rural, Remote

Lead is ubiquitous in nature and even soil in the most remote locations will contain some quantity of lead. In overburden on a near-surface lead ore deposit, soil lead concentrations will be higher than in non-mineralized areas. Lead in the C horizon above a lead sulphide deposit in New Brunswick was 1431 ppm, while in a C horizon removed from such a deposit, the concentration was 65 ppm (Presant and Tupper, 1965, cited in RSC, 1986). In Ontario, there are three base metal mines producing lead as a byproduct (Bigauskas, 1985). It is not known if soil lead concentrations near these deposits have been reported or if the deposits are sufficiently close to the surface to contribute to the soil concentrations.

Soils in rural Ontario locations have been found to contain lead concentrations ranging from 5 to 360 ppm when sampled at depths of 0 to 5 cm (Rinne, 1986). However, sampling locations in this study included areas near roads and thus cannot be considered remote (Rinne, 1988). It is difficult to establish a typical, quantitative value for lead in soil in remote rural locations. The mineralogy of the soil, and more importantly, the organic matter content, are critical variables affecting the apparent background levels of lead in soil. A review of the literature concluded that background levels of lead in soil are probably less than 50 ppm (de Treville, 1964, 1973).

A data report from the Ontario Baseline Soil Survey contains information on the chemical content of soils collected at undisturbed rural sites (MOEE, 1985b; 1985c; 1985d). The report does not include an interpretation or summary of the data. However, the reported lead

concentrations are generally less than 20 ppm in the mineral horizons. Higher levels are usually associated with organic surface horizons in forested sites, but are still less than 100 ppm. Atmospheric sources may contribute to the surface burden of lead, but chelation by organic acids and the low bulk density of the organic horizons are believed to be the cause of the higher lead concentrations.

D-3.2.2 Lead in Soil - Rural, Agricultural

Soil management practices, including agriculture, may contaminate native soil. Application of limestone, phosphate fertilizer, sewage sludge or peat may elevate soil lead concentrations (Lagerwerff, 1967, cited in NRCC, 1973). The Ontario Guideline for Sewage Sludge Utilization on Agricultural Lands only permits sewage sludge application on soils with less than 60 ppm lead (MAF, MOEE & MOH, 1986).

Historical use of lead arsenate as a pesticide has contributed to soil contamination in orchards. Levels as high as 360 ppm have been reported in Nova Scotia orchard soil as opposed to a maximum of 73 ppm in non-orchard soil (Chisholm and Bishop, 1967).

An extensive survey of agricultural soil in Ontario has been carried out in which 296 fields were sampled to a depth of 15 cm and the soil analyzed for a range of metals, including lead (Table D-3-1). Many of the fields were known to have been contaminated by lead arsenate. The background levels for uncontaminated soil was estimated to range between 4.6 and 23.6 ppm (Frank *et al.*, 1976).

The Holland Marsh area in southern Ontario is a productive agricultural area used almost exclusively for vegetable crops. Lead concentrations at the surface of these highly organic soils were 22 ppm, decreasing to 10 ppm at a depth of 48 cm. Mineral soils in the area contained about 2 ppm lead (Czuba and Hutchinson, 1980).

A survey of Essex County found that, excluding urban areas and roadsides, the mean soil lead concentration was 12 ppm (Weis and Barclay, 1984).

D-3.2.3 Lead in Soil - Urban, Residential

There is no "typical" urban residential site. Urban residential sites may range from towns without any industrial activity to a heavily trafficked metropolis with several industries, including industries refining or using lead. Thus, the term "urban residential" will be used to apply to built-up areas that are not obviously associated with a lead-related industry. Such areas, however, may still be affected by industry or vehicle exhaust.

Urban residential areas may have soil lead levels of a few hundred parts per million in the absence of proximal industry. A survey was conducted of a downtown area near an expressway. The area was to serve as a control for samples collected near lead industries. Levels of lead in the top 0 to 5 cm soil averaged 482 ppm with a range of 18 to 1450 ppm (Linzon, 1976).

Based on extensive soil lead data for urban areas, the Ministry of the Environment has set 500 ppm as the "upper limit of normal" for soil lead in urban areas (MOEE, 1986c). The high range in the data probably reflects the constant manipulation of soil conditions. For instance, sodding of lawns, tilling or application of more fertile soil to garden plots would add relatively uncontaminated soil from rural areas.

TABLE D-3-1 LEAD CONTENT OF AGRICULTURAL SOILS IN ONTARIO

AGRICULTURAL PRACTICE AND SOIL TYPE	LEAD CONCENTRATION (ppm dry weight)			
	NUMBER OF SAMPLES	MEAN	RANGE	STANDARD DEVIATION
<u>CROP PRODUCTION</u>				
General crops				
Unimproved	15	12.5	3.2-33.7	7.5
Field Crops	126	11.4	2.3-47.9	4.1
Vegetables				
Mineral Soils	56	13.3	1.5-50.1	10.2
Organic Soils	13	12.6	2.3-42.2	11.0
Fruit				
Apples	31	247.0	6.4-888.0	207.0
Sweet Cherries	16	109.0	11.6-233.0	69.1
Sour Cherries	12	71.2	4.4-235.0	80.1
Peaches	11	26.4	6.0-69.0	19.2
Grapes	16	19.1	8.0-35.5	7.9
Total and Mean				
Tree and Vine Fruit	86	123.0	4.4-888.0	165.0
Other Crops	210	14.1	1.5-50.1	9.5
<u>SOIL TYPES</u>				
Organic soils	13	12.6	1.5-50.1	11.0
Sandy soils				
Fruit	43	129.4	4.4-888.0	178.4
Other Crops	82	10.4	2.3-47.5	8.4
Loam soil				
Fruit	29	153.3	6.0-654.0	171.7
Other Crops	69	17.6	4.0-49.3	9.9
Clay soil				
Fruit	14	43.5	8.0-208.0	59.0
Other Crops	46	15.7	1.5-50.1	15.8
Total and Mean	296	45.8	1.5-888.0	102.0

Source: Frank *et al.*, 1976.

D-3.2.3.1 Lead-based Paint

Lead-based paint has been identified as a source of high exposure and as a cause of lead poisoning in children in certain areas of the United States. In 1989, as a result of complaint investigations, the Phytotoxicology Section of the Ministry reviewed eight properties in the Metropolitan Toronto area (Table D-3-2). They were identified as potentially contaminated with old lead-based paint. Soil samples were taken at sites both near to and away from the wall with peeling paint. All but one site had soil lead concentrations within the upper limit of normal, or the Ontario urban background level of 500 ppm⁷. This house was poorly maintained and located in the vicinity of Canada Metal Company, a secondary lead smelter.

At each site, the soil lead concentration decreased with increasing distance from the structure, suggesting a correlation between soil lead concentrations and lead-based paint. The measured concentrations of soil lead ranged from 43 to 1050 ppm (dry weight) (MOEE, unpublished data).

Samples of flaking paint were also taken to determine the lead concentrations at the sites. The samples were categorized according to colour so that the lead concentration is based on the colour of paint (Table D-3-3).

In another study, paint samples were taken from a site in Elmira, Ontario, where storage tanks were kept. Lead levels ranged from 21 ppm (yellow paint) to 350,000 ppm (dark green) (Phytotoxicology Section, MOEE, unpublished data, 1989).

The ULN guidelines are defined as the mean of the available information plus 3 standard deviations. These ULN's are not segregated into landuse categories.

TABLE D-3-2 MEAN SOIL LEAD CONCENTRATIONS NEAR HOUSES WITH LEAD-BASED PAINT SURFACES

PROPERTY NUMBER	SAMPLING SITE LOCATION NEAR (0.5 m) FAR (3 m)	SOIL LEAD CONCENTRATION (ppm, dry weight)
1	near	245
	far	162
2	near	295
	far	225
3	near	350
	far	175
4	near	260
	far	124
5	near	63
	far	43
6	near	1050
	far	215
7	near	265
	far	113
8	near	355
	far	145

TABLE D-3-3 LEAD CONCENTRATIONS FROM EXTERNAL COLOURED PAINT, METROPOLITAN TORONTO

PAINT COLOUR	LEAD CONCENTRATION (ppm, dry weight)
White	1100 to 290,000
Green	164,000 to 330,000
Gray	34,000 to 230,000
Brown	2500 to 330,000
Pink	250,000
Beige	45,000
Blue	44,000
Red	4900 to 54,000

D-3.2.4 Lead in Soil - Urban, Roadside

Emissions from vehicles burning leaded fuels result in elevated lead levels in the soil near roads. Rates of lead accumulation depend on traffic density and the proportion of vehicles using leaded fuel. Comprehensive investigations of the relationship between soil lead, traffic volume and distance from the road are not available for Ontario.

A number of reports are available on the U.S.A. In Illinois, soil samples 0 to 10 cm deep were collected along transects from roads with different traffic densities. Traffic density was a factor. For samples taken at a distance of five meters from the road, soil lead was 93 ppm for a road with 8100 vehicles per day and 24 ppm for one with 550 vehicles per day. Soil lead levels declined rapidly with distance from the roads. At the 8100 vehicle-per-day site, the soil lead concentration was 1225 ppm at 0.3 meters from the road, decreasing to 526 ppm at 1 meter and 13 ppm at 100 meters. It was concluded that the vehicle-derived lead demonstrated a dual particle size distribution, with the larger particles settling out within 5 meters and the smaller particles within 100 meters (Wheeler and Rolfe, 1979).

It is reasonable to assume that similar distributions and soil lead levels would occur near Ontario roads. A mean soil lead concentration of 292 ppm (0 to 2.5 cm deep) was found for samples from 65 major intersections in Toronto. However, distances from roads or traffic volumes were not specified (Linzon *et al.*, 1976). A survey of soil (0 to 10 cm deep) at 11 locations in Hamilton revealed lead concentrations ranging from 63 to 373 ppm. Once again, traffic volumes and distances from the road were not given, although the presence of lead was ascribed to vehicular emissions (Temple, 1974).

D-3.2.5 Lead in Soil - Urban, Industrial

In any industrial area, certain industries are likely to be using lead. These may be auto body repair shops, battery manufacturers or large scale refining operations. Emissions from such industries will depend on the amounts of lead used or produced; the processes involved; and the controls in place. Isolating the contributions from lesser industries is difficult. However, an industry heavily involved with lead can have a marked effect on the already elevated soil lead levels found in an urban environment. Two such industrial areas are located in Toronto and a third in Mississauga.

Soil lead data from samples obtained in the vicinity of the three companies are summarized in Table D-3-4 (MOEE, 1986d; 1986e; 1986f). Samples were obtained within 1000 meters of the source in most cases. The data do not reflect the extreme range in lead concentrations. The lowest level reported was 28 ppm (near the Canada Metal Company) while the highest was 51,000 ppm (near Toronto Refiners and Smelters). The variation reflects the activity of home owners and others in improving the properties through soil replacement and fresh sodding.

TABLE D-3-4 LEAD CONCENTRATIONS IN SOIL NEAR THREE SECONDARY LEAD SMELTERS IN TORONTO AND MISSISSAUGA, ONTARIO

COMPANY	NUMBER OF SAMPLE SITES	MEAN SOIL LEAD CONCENTRATION (ppm)		
		1980 (0-5 cm)*	1983 (0-1 cm)*	1985 (0-5 cm)*
Toronto Refiners and Smelters	11	5190	7967	11745
Canada Metal Company	24	1584	1130	800
Tonolli Co. of Canada	41	1078	1894	1230

* Depth of soil sample.

Source: MOEE, 1986d; 1986e; 1986f

In 1985, 91% of the stations around Toronto Refiners and Smelters exceeded the 500 ppm "upper limit of normal". Corresponding exceedances were 43% and 34% for the Canada Metal Company and Tonolli, respectively.

Historical contamination from industries no longer in operation can still be a cause for concern for contemporary land use. One such site is the Davis Tannery Property, in Kingston, Ontario. Proposals for residential and recreational development in the late 1970's raised concern over residual lead contamination from a lead smelter that had ceased operation in 1916. Soil samples from 11 sites showed a mean lead concentration at the surface of 2252.1 µg/g (range: 174.0 to 11,666.7 µg/g), and a mean of 789.7 µg/g (range: 106.5 to 3208.3 µg/g) at a depth of 5 cm (Stokes, 1977).

D-3.3 Levels of Lead in Terrestrial Biota

Lead is ubiquitous in the terrestrial environment, so that all terrestrial organisms are exposed to some extent. Levels of lead in terrestrial animals indicate the magnitude and scope of environmental contamination, and can illustrate the movement of lead along the food chain.

D-3.3.1 Lead Levels in Terrestrial Animals

D-3.3.1.1 Microorganisms and Invertebrates

No data could be found on lead concentrations in microorganisms and invertebrates in Ontario. Data from elsewhere, however, were found for earthworms and insects.

Earthworms can have lead concentrations approximating or surpassing that of the soil. Earthworms along highways in Maryland, U.S.A., living in soil ranging from 34.9 to 700 ppm lead, accumulated concentrations of 38.5 to 331.4 ppm dry weight.

Levels in the worms mirrored the pattern in the soil, being higher along more heavily-travelled roads (Gish and Christensen, 1973). Earthworms from a contaminated site in Wales were found to have lead levels one to four times the soil lead concentration (Ireland, 1975; Ireland and Richards, 1977).

Earthworms from plots experimentally amended with sewage sludge and fertilizer had lead concentrations that were correlated with soil concentrations, but were not concentrated (Beyer *et al.*, 1982; Kruse and Barrett, 1985). The average lead concentrations in worms from soil treated with sewage sludge were three times those from fertilizer-treated or control plots. All were well below the concentrations found in worms from roadside soil (Gish and Christensen, 1973) and in worms near a lead/zinc mine (Ireland, 1975; Kruse and Barrett, 1985).

Predatory insects tend to have higher lead concentrations than herbivorous insects. Among the latter, chewing insects have higher concentrations than sucking insects. This difference was found to be especially pronounced in areas where superficial lead deposition was greatest, such as along highways (Wade *et al.*, 1980).

D-3.3.1.2 Wildlife

The National Academy of Sciences estimates that the "natural" levels of lead in the bones of herbivores range between 0.04 and 0.12 µg/g dry weight. Levels for carnivores range from 0.01 to 0.03 µg/g dry weight (NAS, 1980). Wild animals are exposed through indirect sources, such as contaminated food, water and air, or direct sources, such as lead shot. The latter is the largest source of lead poisoning in wildlife.

The levels of lead in wildlife are indicative of the overall lead contamination of their environment. A review of lead hazards to wildlife found that lead concentrations tended to be higher in birds and mammals from urban locations, and from those near metal mines, smelters, and plants that reclaim storage batteries (Eisler, 1988). Published data on lead levels in wildlife are few, but some are available for Ontario. Much of the research has been conducted on birds of the Great Lakes. A recent study measured lead concentrations in herring gulls (*Larus argentatus*) from four of the lakes (Table D-3-5).

TABLE D-3-5 LEAD CONCENTRATIONS IN COMPOSITE TISSUE SAMPLES FROM ADULT HERRING GULLS IN THE GREAT LAKES

LOCATION	LEAD TISSUE CONCENTRATIONS (µg/g) (wet weight)				
	NUMBER OF SAMPLES	LIVER	KIDNEY	FEATHER	BONE*
Lake Ontario: Hamilton Harbour	9	0.13	6.90	7.90	15.0 (17.5)
Lake Erie: Middle Island	10	0.05	0.06	1.93	2.5 (2.95)
Lake Huron: Double Island	13	0.20	0.69	3.86	30.0 (36.8)
Lake Superior: Agawa Rock	15	0.27	0.93	4.75	17.0 (20.7)
MEAN		0.16	2.15	4.61	16.1 (19.5)

* g/g dry weight values in brackets

Source: Struger *et al.*, 1987.

The "normal" background levels of lead in birds are believed to range from 2 to 15 µg/g dry weight in bone; from 1 to 10 µg/g dry weight in kidney; and 0.5 to 5 µg/g dry weight in liver (Connors *et al.*, 1975; Kendall and Scanlon, 1981; Custer *et al.*, 1984, all cited in Scheuhammer, 1987).

The liver and bone data indicate that gulls in the upper Great Lakes are exposed to relatively substantial sources of lead, probably from their food supply. The latter can include insects, earthworms, small mammals, songbirds and garbage when fish are scarce (Allan, 1978). Concentrations in the livers of birds from all four lakes were below the level considered to be diagnostic of lead poisoning in mallard ducks (*Anas platyrhynchos*) (Longcore *et al.*, 1974). However, lead levels in the kidneys of gulls from Hamilton Harbour may be indicative of lead poisoning (Struger *et al.*, 1987).

Lead concentrations in common terns (*Sterna hirundo*) from Lake Ontario and coastal Massachusetts have been investigated, and are presented in Table D-3-6.

Common terns from Hamilton Harbour seem to have lower lead levels in their bones than terns from Great Gull Island. Lead levels in the bones of common terns from Hamilton Harbour are also lower than in the bones of herring gulls from the same site (albeit twelve years earlier). The remaining data are reported as dry weight concentrations and thus cannot be compared to the wet weight concentrations in herring gulls (Table D-3-5).

TABLE D-3-6 LEAD CONCENTRATIONS IN TISSUES OF ADULT COMMON TERNS FROM GREAT GULL ISLAND, MASSACHUSETTS AND HAMILTON HARBOUR, ONTARIO

TISSUE TYPE	LEAD CONCENTRATIONS IN COMMON TERNS ($\mu\text{g/g}$) (dry weight)	
	GREAT GULL ISLAND	HAMILTON HARBOUR
Bone		
Mean	18.1	12.2
Range	11.2-32.8	9.7-15.9
Breast		
Mean	<1.0	<1.0
Liver		
Mean	<10.0	<10.0
Kidney		
Mean	<10.0	<10.0

* Probability that the distribution of lead is equivalent in the two populations.
Data from Connors *et al.*, 1975.

Lead levels in birds in the United States vary with the species and location. A nation-wide survey of levels in the wingbones of waterfowl found lead concentrations averaging between 2 and 48 $\mu\text{g/g}$ dry weight (Stendell *et al.*, 1979). The results are similar to the average lead concentrations of 2 and 24 $\mu\text{g/g}$ dry weight found in wing bones of waterfowl from Chesapeake Bay, U.S. (DiGiulio and Scanlon, 1984).

House sparrows (*Passer domesticus*) and robins (*Turdus migratoris*) from urban areas had lead levels in their femurs averaging as high as 130 and 133 $\mu\text{g/g}$ dry weight, respectively (Getz *et al.*, 1977a). Concentrations in the femurs of rural house sparrows and robins from this same study averaged 17 and 41 $\mu\text{g/g}$ dry weight, respectively. Ruffed grouse (*Bonasa umbellus*) in Virginia were found to have lead concentrations in their bones ranging from 0.4 to 9 $\mu\text{g/g}$ dry weight (Kendall *et al.*, 1984). Research on the sora rail (*Parzana carolina*) in Maryland found levels in bone ranging from <0.4 to 127 $\mu\text{g/g}$ dry weight. The higher concentrations were in birds found to have lead shot in their gizzards (Stendell *et al.*, 1980, cited Eisler, 1988).

Lead levels in mammals greatly depend on location. Levels in the bones of field mice (*Apodemus sylvaticus*) ranged from 189 and 352 $\mu\text{g/g}$ dry weight near an abandoned lead mine, compared to 11 to 21 $\mu\text{g/g}$ dry weight at a control site (Roberts *et al.*, 1978). Average lead concentrations in the bones of white-tailed deer (*Odocoileus virginianus*) near a zinc smelter were 9 $\mu\text{g/g}$ dry weight; 100 km from the smelter concentrations were 6 $\mu\text{g/g}$ dry weight (Sileo and Beyer, 1985).

Urban wildlife can be useful indicators of lead contamination within, and between, cities. Short-tailed shrews (*Blarina brevicauda*) and deer mice (*Peromyscus maniculatus*) had bone lead concentrations of 67 and 52 µg/g dry weight, respectively, in areas with high traffic densities. Bone lead concentrations in shrews and mice from areas of low density traffic were 12 and 5 µg/g dry weight, respectively (Getz *et. al.*, 1977b; Mierau and Favara, 1975). Levels of lead in the bones of urban commensal rats (*Rattus norvegicus*) averaged 125 µg/g fresh weight, compared to 8 µg/g in their rural cohorts (Way and Schroder, 1982). The ability of the eastern gray squirrel (*Sciurus carolinensis*) to thrive in large urban centres like Montreal and Toronto, as well as in remote natural habitats, makes it an ideal candidate for research. Several surveys of the lead levels in squirrels have been done and the data are summarized in Table D-3-7.

TABLE D-3-7 CONCENTRATIONS OF LEAD IN TISSUES OF GRAY SQUIRRELS IN TORONTO, OHIO AND FLORIDA

SITE	MEAN LEAD CONCENTRATION ± STANDARD DEVIATION (µg/g wet weight)	
	KIDNEY (NUMBER OF SAMPLES)	LIVER (NUMBER OF SAMPLES)
Ontario: [*]		
Toronto	1.7 ± 1.2(10)	1.8 ± 1.3(10)
Mississauga and Uxbridge	0.7 ± 0.8(29)	0.5 ± 0.6(28)
Upland Ohio ^{**}	0.5 ± 0.0(15)	2.0 ± 1.2(4)
Florida: ^{***}		
Jacksonville Wildlife Management Area	1.2 ± 1.4(20) 0.3 ± 0.1(7)	— —

* Source: Murray, 1987.

** Gray squirrels and fox squirrels (*Sciurus niger*). Source: Lynch, 1973.

*** Source: McKinnon *et al.*, 1976.

In spite of the limited data, lead levels in Toronto squirrels seem to be comparable to those in urban squirrels in Florida. Levels in squirrels from suburban Mississauga and rural Uxbridge are slightly higher than those from the forests of Ohio and a Florida wildlife area.

Marine mammals are at the top of the marine food chain. Lead levels in marine mammals are therefore important indicators of aquatic lead contamination. Such lead levels are also of concern to people harvesting these animals for food. In a compilation of data on heavy

metals in marine mammals from northern waters (Wagemann and Muir, 1984), mean lead concentrations in the liver tissue of ringed seals (*Phoca hispida*) in the Canadian eastern arctic in 1975, 1976 and 1977 were <0.03, <0.04, and <0.05 µg/g, respectively (Fallis, unpublished data).

These values were similar to those found in the liver tissue of Ringed Seals in western Greenland (Johansen *et al.*, 1980). In contrast, mean lead concentrations in the liver tissue of Harbour Seals (*Phoca vitulina*) from the German North Sea coast between 1974 and 1976 ranged from 0.17 to 0.45 µg/g (Drescher *et al.*, 1977).

D-3.3.1.3 Domestic Animals

Domestic animals are exposed to several direct and indirect sources of lead. Lead poisoning in cattle, horses and swine has been caused by: lead-based paint, used motor oil, grease, used batteries, linoleum, putty, roofing material, contaminated water, contaminated feed, and pastures contaminated by lead smelters (U.S.EPA, 1977). As with wildlife, there are few data on lead levels in domestic animals.

Lead concentrations in roadside grass decline rapidly with distance from the road. Therefore, domestic animals would have to routinely graze near roads for vehicular lead to be a significant source. Surveys in Ontario were not available.

Concentrations of lead in domestic slaughter animals are of particular interest for human health. Such levels were investigated in Saskatchewan in the mid-1970's. All of the reported values were below the then Canadian official tolerance level of 2 ppm. More recent data on domestic slaughter animals are not available.

Domestic dogs in an urban environment are exposed to many of the same sources of lead as young children: they breathe the same air, stir up the same dust, play in the same dirt, and sometimes even eat the same food. Sources of lead poisoning in dogs include lead-based paint, linoleum, curtain weights, and plaster (U.S.EPA, 1977). The same items are often inadvertently consumed by young children, a behaviour known as "pica".

Dogs can be useful indicators of situations presenting a risk of lead poisoning for children. Surveys of lead in the blood of dogs could be a less costly alternative to large-scale human surveys. In Winnipeg, a survey of blood lead concentrations in dogs found levels to be similar to those in children. In addition, dogs from areas of heavy traffic had higher blood lead concentrations than did dogs from areas with less traffic (Table D-3-8) (Kucera, 1983).

TABLE D-3-8 BLOOD LEAD CONCENTRATIONS OF DOGS LIVING WITHIN 1000 METRES OF WINNIPEG STREETS

VEHICLE-PASSES WITHIN 1000m, (thousands/12-hour day)	NUMBER OF SAMPLES	MEAN BLOOD LEAD CONCENTRATION (µg/dL)
< 2	3	1.97
> 2 - 5	28	2.48
> 5 - 10	98	3.06
> 10 - 20	152	2.72
> 20 - 30	59	3.07
> 30 - 40	21	3.58
> 40 - 50	8	4.96
> 50	4	10.49

Source: Kucera, 1983.

D-3.3.2 Levels of Lead in Vegetation

There are numerous reports on accumulation of lead in plants as a function of lead in the growing medium. These experiments range from exposing crop plants to lead in a hydroponic solution (John, 1977) to amending natural soil with lead salts (Baumhardt and Welch, 1972; Chisholm, 1972) or with lead-containing sewage sludges (Keefer *et al.*, 1986).

Many investigators have shown that plants can accumulate lead from soil, or from direct application to foliage and stems. Conflicting results have been reported in the literature with respect to: the amounts lead absorbed; where in the plant accumulation occurs; and to what degree translocation occurs. Some workers believe that the lead content of plants is highly correlated with the lead content in soil (Kovalevskiy, 1979; Bisessar and McIlveen, 1991c) while others believe that no simple relationship exists.

Lead uptake by aquatic and terrestrial plants is influenced by environmental factors, such as geochemical anomalies, pollution, and seasonal variation. However, the sediment/soil adsorption characteristics of the growing medium are the determining factors, because lead must be biologically available for uptake to occur. For example, a high organic content will reduce lead uptake because lead tends to bind to organic matter. Millar showed that lead accumulation in corn depended on the level of lead in the soil relative to the soil's capacity to adsorb lead (Millar *et al.*, 1975).

Environmental factors may also influence the type of plant tissue that will accumulate lead. For instance, increasing the pH of lead-contaminated soils appears to decrease foliar lead but

has little effect on lead in roots (Zimdahl and Foster, 1976). Most soil lead is unavailable to plant roots because it is not readily soluble. However, soluble forms present in nutrient solutions may be taken up in great amounts (Cannon, 1976; Zimdahl and Koeppe, 1977). Lead is absorbed mainly by root hairs and is stored to a considerable degree in cell walls.

There is evidence to suggest that the lead measured in plant tissue is passively absorbed (Zimdahl, 1975; Hughes, 1980). Histological analysis of *mays* exposed to different solutions of lead showed that the roots initially accumulated a surface precipitate of lead salts. Lead was slowly taken into the roots where it appeared as much smaller crystals encased in vesicles, primarily associated with the cell walls (Malone *et al.*, 1974). Lead may also form insoluble complexes with cell wall constituents. Although some of these vesicles slowly migrated to other tissues, most of the lead remained in the roots.

Evidence suggests that in some species of terrestrial plants, soil lead is not readily translocated from roots to above ground tissue (Zimdahl and Koeppe, 1977; Zimdahl *et al.*, 1978). Studies of different plants have found that roots generally accumulate lead to a greater degree than shoots (Motto *et al.*, 1970; Jones and Hatch, 1945). For example, one study found that only 3% of lead from the root was translocated to the shoot (Zimdahl, 1975). Variation with the plant species was demonstrated in beets and lettuce grown in lead-contaminated soil. At some soil lead concentrations, foliar accumulation of lead was greater than the root content (Table D-3-9) (Bisessar and McIlveen, 1991a).

Of greatest concern is the amount of lead present in the edible portions of crops. Although lead generally accumulates in the roots, it may be present in the above ground tissues (Bisessar and McIlveen, 1991a, 1991b, 1991c). Beet foliage stored more lead than beet roots. Lettuce foliage had more lead than that measured in both beet roots and shoots (Table D-3-10) (Hutchinson *et al.*, 1974). Thus, the distribution of lead accumulation varies with the species and it cannot be assumed that root crops will be the only significant source.

Estimates of biouptake in plants are needed in order to determine the relative exposure to lead from vegetation. Biouptake depends the bioavailability of lead in the growing medium and on the species. Figures D-3-1 and D-3-2 show the relationship between soil lead and lead in lettuce and beets, and, cabbage and carrot (Bisessar and McIlveen, 1991a, 1991b, 1991c). This allows calculation of the uptake factors as follows:

SELECTED CROP	UPTAKE FACTOR, µg/g
carrot root	0.02(2%)
beet root	0.0067(1%)
beet foliage	0.0156(2%)
lettuce foliage	0.076(8%)
cabbage shoot	0.0025(0.25%)

There is some evidence of foliar absorption of lead due to atmospheric deposition of dust and particulate. As with the uptake of lead from soil, accumulation of lead through direct foliar application varies with the species and environmental conditions, such as aerosol properties, leaf surface characteristics and meteorological characteristics (Dollard 1986; WHO 1989; Palmer and Kucera, 1980; Koslow *et al*, 1977). A leaf with a pubescent surface will be more apt to retain particulate on its surface long enough for it to be absorbed into the tissue. Waxy leaf surfaces do not allow for great retention therefore less is absorbed (Page *et al*, 1971; Wedding *et al*, 1975).

TABLE D-3-9 LEAD ACCUMULATION IN LETTUCE AND BEETS

SOIL Pb ($\mu\text{g/g}$)	LETTUCE		BEET	
GREENHOUSE	ROOT	SHOOT	ROOT	SHOOT
54	32	5	6	6
501	698	35	10	26
1566	1517	103	27	34
3483	1983	162	29	39
FIELD				
16	10	4	1	<1
923	131	92	5	15
1440	171	144	8	19
3140	323	314	15	47

Source: Adapted from Bisessar and McIlveen, 1991c (dry weight)

It is difficult to measure lead uptake through foliage as a proportion of that deposited because the latter may be washed away by rain. Direct application of radioactively labelled lead nitrate to the leaves of lettuce and radish indicated some absorption into the leaves and no more than 2% subsequent translocation (Hempjill and Rule, 1975). Another study suggested a possible enhancement of lead absorption when administered to a damaged leaf surface (Dollard, 1986).

In contaminated waters, lead is tightly bound to sediment with little in solution. However, when both submerged and floating plants were exposed to lead nitrate at concentrations of 1 and 10 $\mu\text{mol/L}$ for 41 to 46 and 70 to 73 days, respectively, the floating plants had higher tissue lead concentrations (Werff and Pruyt, 1982). Floating plants may have had added exposure through atmospheric deposition of particulate. Dose-related increases in different plant tissues have been observed upon exposure to dissolved lead. Accumulation was greatest in the roots, followed by stems, then leaves (Hay *et al*, 1984). With some species of

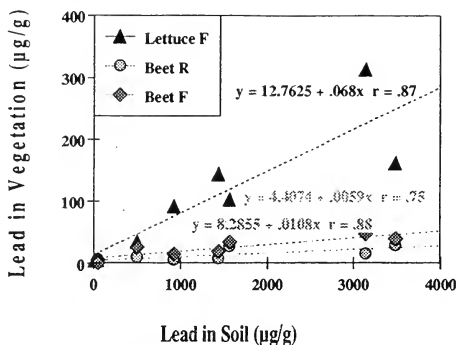


Figure D-3-1 Relationship between soil and plant lead levels in lettuce (foliage) and beets (roots, foliage)

Source: Bisessar and McIlveen, 1991c

plants lead accumulation may be affected by seasonal changes (Kay *et al*, 1984).

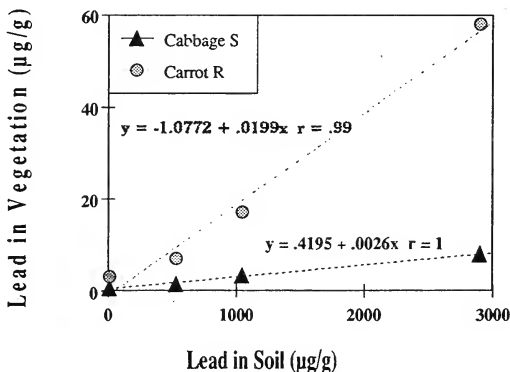


Figure D-3-2 Relationship between soil and plant lead levels in cabbage (shoot) and carrot (root)

Source: Bisessar and McIlveen, 1991b

D-3.3.2.1 Food Products from Agricultural Areas

In Ontario, the dry weight concentrations of lead in corn and soybean seed in Essex County averaged 3.77 ppm (N = 8, SE±2.19) and 0.96 ppm (N = 15, SE±0.12), respectively (Weis and Barclay, 1984). Another study analyzed lead concentrations in a variety of vegetables grown on the Holland Marsh. The data for edible plant parts sampled in the fall or at the time of harvest are summarized in Table D-3-10 (Czuba and Hutchinson, 1980).

The lead concentrations of crops grown in experimental field plots in Nova Scotia have been reported. Two soil types were used with different lead concentrations. Lead arsenate was applied to the soils to elevate the lead concentration. Table D-3-11 is taken from this report; wet and dry weight concentrations are provided for comparison with Table D-3-12 (Chisolm, 1972). The above data are comparable to those of U.S. researchers (Wolnik *et al.*, 1983; 1985). Normal "background" concentrations of lead were derived by examining raw agricultural crops in major U.S. growing areas, where human activities are limited to agricultural practices.

TABLE D-3-10 LEAD CONCENTRATIONS IN CROPS GROWN ON THE HOLLAND MARSH, ONTARIO

CROP	LEAD CONCENTRATION (ppm, dry weight)		
	N	MEAN	SD
Lettuce leaf	23	7.2	± 2.6
Celery leaf	24	11.5	± 3.6
Potato tuber	12	4.4	± 0.9
Carrot root	44	4.4	± 1.6
Onion leaf	36	9.9	± 1.9
Onion bulb	19	4.9	not given
Cauliflower flower	12	40.2	± 1.1
Cabbage leaf	12	7.3	± 3.4

Source: Czuba and Hutchinson, 1980

TABLE D-3-11

LEAD LEVELS IN PLANTS GROWN ON LEAD ARSENATE-TREATED AND UNTREATED SOD IN NOVA SCOTIA

YEAR	SITE	PLANT PART	LEAD CONCENTRATION (DRY WEIGHT, ppm)		LEAD CONCENTRATION (WET WEIGHT, ppm)	
			UNTREATED*	TREATED**	UNTREATED	TREATED
1967	A	Beet tops	8.41	20.77 ± 2.52	0.90	2.26 ± 0.27
		Beet roots	2.68	14.73 ± 0.74	0.44	2.44 ± 0.13
		Onion tops	6.14	21.74 ± 2.65	0.92	3.26 ± 0.04
		Onion bulbs	1.00	1.90 ± 0.14	0.15	0.29 ± 0.02
		Swiss chard	5.43	6.39 ± 0.82	0.63	0.77 ± 0.09
1968	A	Lettuce	1.73	5.23 ± 0.30	0.25	0.76 ± 0.04
		Green beans	1.97	2.95 ± 0.30	0.28	0.43 ± 0.04
		Carrot roots	1.60	6.58 ± 0.43	0.20	0.82 ± 0.05
1969	A	Corn kernels	4.45	15.04 ± 0.78	0.57	1.93 ± 0.01
		Green beans	1.12	1.11 ± 0.06	0.28	0.28 ± 0.01
1968	B	Beet roots	2.31	4.50 ± 2.34	0.38	0.74 ± 0.38
		Turnip roots	1.25	1.85 ± 0.23	0.20	0.30 ± 0.03
		Carrot roots	1.61	5.31 ± 0.53	0.17	0.56 ± 0.05
		Parsnip roots	0.70	6.88 ± 0.86	0.15	1.49 ± 0.17
1969	B	Corn kernels	4.56	18.05 ± 4.56	0.48	1.90 ± 0.47
		Green beans	0.68	0.97 ± 0.09	0.17	0.24 ± 0.02

* single determination ** mean and standard error of 4 replicates

Source: Chisholm, 1972

Notes:

1. Site A soil: untreated, 37.5±4.8 ppm Pb; treated, 277.5±12.3 ppm Pb
2. Site B soil: untreated, 13.1±0.6 ppm Pb; treated, 145.0±13.5 ppm Pb

TABLE D-3-12 BACKGROUND LEAD CONCENTRATIONS IN CROPS GROWN THROUGHOUT THE UNITED STATES

CROP	N	MEAN LEAD CONC., WET WEIGHT, ppm (DRY WEIGHT)	RANGE	%RSD*
Carrots	207	0.009(0.079)	0.001-0.125	127.5
Field corn	277	0.022(0.026)	<0.002-2.75	791.5
Onions	230	0.005(0.049)	<0.0002-0.054	106.8
Rice	166	0.007(0.008)	<0.002-0.070	114.4
Spinach	104	0.045(0.584)	0.016-0.17	52.2
Tomatoes	231	0.002(0.032)	<0.0001-0.025	121.2
Lettuce	150	0.013(0.313)	0.001-0.078	112.7
Peanuts	320	0.010(0.011)	<0.007-0.194	143.1
Potatoes	297	0.009(0.047)	0.0002-0.370	292.6
Soybeans	322	0.042(0.046)	<0.0016-0.323	88.6
Sweet corn	268	0.003(0.015)	<0.0002-0.034	126.1
Wheat	288	0.037(0.042)	<0.0008-0.716	168.5

* percent relative standard deviation

Source: Wolnik *et al.*, 1983; 1985

D-3.3.2.2 Food Products Near Point Sources of Lead

Of specific interest are those food crops grown in soil contaminated by industrial or vehicular emissions. A study was done in New Jersey on the lead concentrations in vegetable crops grown in soil contaminated by vehicle emissions (Table D-3-13) (Motto *et al.*, 1970). The values were generally an order of magnitude greater than the control (untreated) values (Table D-3-12).

TABLE D-3-13 LEAD CONTENT (PPM) OF FIVE CROPS GROWN AT THREE FIELD SITES IN NEW JERSEY

TRAFFIC VOLUME	12,500/24 HRS.			47,100/24 HRS.			49,000/24 HRS.		
DISTANCE FROM HIGHWAY (ft)	30	100	250	30	100	250	30	100	250
Carrot									
tops	18	11	14	37	26	21	53	22	17
roots	3.8	5.3	3.9	6.2	9.5	9.4	9.1	10	5.0
Corn									
tassel	31	7.4	7.8	179	144	69	--	--	--
leaves	19	17	14	86	47	36	88	51	40
stalk	3.6	3.7	0.9	5.6	3.6	0.2	6.2	3.4	3.6
husk	--	--	--	3.0	5.0	2.6	--	--	--
roots	6.0	3.9	5.4	19	14	19	54	19	--
kernel	3.8	3.6	3.1	0.0	0.2	0.2	--	--	--
cob	8.0	3.2	2.6	0.4	0.0	0.4	--	--	--
Lettuce									
leaves	12	13	--	24	21	14	56	35	--
roots	16	15	--	24	27	w	61	32	--
Potato									
leaves	36	31	21	87	47	29	--	--	--
stems	12	8.4	7.8	15	11	14	--	--	--
roots	22	23	18	33	49	58	--	--	--
tuber	0.5	1.5	1.0	2.6	3.0	3.0	--	--	--
Tomato									
leaves	36	25	17	76	82	40	88	52	44
stem	9.0	9.8	6.9	27	25	31	29	13	7.7
root	11	15	14	27	35	50	37	12	9.6
fruit	2.8	3.0	2.4	4.6	2.7	2.8	3.6	1.2	3.2

* Wet weight or dry weight not specified

Source: Motto *et al.*, 1970

In Ontario, significantly higher lead concentrations were reported in various vegetables and herbs grown in soils taken from a location near a smelter (Tables D-3-14, D-3-15, D-3-16) (Bisessar and McIlveen, 1991a, 1991b, 1991c).

TABLE D-3-14

LEAD CONCENTRATIONS IN HERBS GROWN IN SOIL TAKEN
IN VICINITY OF SECONDARY LEAD SMELTER

SOIL LEAD CONCENTRATION (ppm)	SAVORY		MARJORAM		BASIL	
	R	S	R	S	R	S
14 (control)	2.2	2.1	3.3	2.4	4.3	1.6
482	36.0	12.0	63.0	20.0	16.0	10.0
797	102.0	80.0	101.0	43.0	40.0	17.0
2500	708.0	299.0	192.0	58.0	107.0	31.0

R=root S=shoot; all lead concentrations, ppm (dry weight)

Source: Bisessar and McIlveen, 1991

TABLE D-3-15

LEAD CONCENTRATIONS IN LETTUCE AND BEETS GROWN IN
SOIL TAKEN IN VICINITY OF LEAD SMELTER ($\mu\text{g/g}$, dry weight)

LETTUCE								
	GREENHOUSE				FIELD			
	54*	501	1566	3483	16*	923	1440	3140
Soil lead, $\mu\text{g/g}$								
Root	32	698	1517	1983	10	131	171	323
Foliage	5	35	103	162	4	92	144	314
BEETS								
	GREENHOUSE				FIELD			
	54*	501	1566	3483	54*	923	1440	3140
Soil lead, $\mu\text{g/g}$								
Root	6	10**	27	29	1	5	8	15
Foliage	6	26	34	39	<1	15	19	47

*control sample

** not significant

Source: Bisessar and McIlveen, 1991

TABLE D-3-16 LEAD CONCENTRATIONS IN CARROTS AND CABBAGE GROWN IN SOIL TAKEN IN VICINITY OF LEAD SMELTER ($\mu\text{g/g}$)

SOIL LEAD CONCENTRATION ($\mu\text{g/g}$)	CARROT		CABBAGE	
	R	S	R	S
16 (control)	3.0	4.0	2.0	0.62
529	7.0	26.0	20.0	1.48
1046	17.0	27.0	50.0	3.32
2900	58.0	151.0	88.0	8.0

R=root S=shoot

Source: Bisessar and McIlveen, 1991b

D-3.3.2.3 Wildlife and Domestic Animal Forage

Domestic animals, if fed the same food crops consumed by humans, would be exposed to the same lead levels. However, if the animals graze in areas contaminated by lead (eg. roadside grass) then the dosage would be reflected by the lead concentration of the forage. Contamination of forage from point sources (lead smelters, old mine sites) can occur, but is not relevant in Ontario.

A significant portion of the diet of members of the deer family consists of lichens. Lichens are considered to be sensitive accumulators of air pollutants since they lack cuticles which exclude pollutants. A survey of lichen (*Cladina rangiferina*) chemistry, which included lead, was recently reported (Zakshak *et al.*, 1986). Samples collected from various locations in Ontario had lead concentrations ranging from 7 to 27 ppm. Generally the concentrations were about 15 to 20 ppm (it is not known if these concentrations are based on wet or dry weight). In contrast, samples from the Northwest Territories averaged less than 5 ppm lead.

The lead content of grass along roadsides has been investigated in New Jersey (Motto *et al.*, 1970). Results are presented in Table D-3-18. In Ontario, investigations have been done on the lead concentrations in certain garden crops grown near two secondary smelters in Toronto compared to an urban control area (Table D-3-17) (Roberts *et al.*, 1974).

TABLE D-3-17

**LEAD CONTAMINATION IN EDIBLE PARTS OF VEGETABLE
CROPS GROWN NEAR A SECONDARY LEAD SMELTER,
TORONTO, ONTARIO**

	100 m NORTH OF SMELTER		300 m EAST OF SMELTER		CONTROL AREA	
	Unwashed	Washed	Unwashed	Washed	Unwashed	Washed
Lettuce leaves	1,506	596	92	27	23	19
Radish tubers	—	169	—	7.1	—	2.5
Tomato fruit	8.5	6	6.2	6.5	3.6	4.2
Soil	8,500		355		85	

All lead concentrations, dry weight, ppm

Source: Roberts *et al.*, 1974

TABLE D-3-18

**LEAD CONCENTRATIONS IN ROADSIDE GRASS SAMPLES IN
NEW JERSEY**

	TRAFFIC VOLUME (Vehicles/24 hours, in thousands)									
	12.8	14.7	17.7	19.7	41.0	45.6	48.6	48.6	54.7	AVE.
DIST. FROM HWY (ft)	NOT WASHED									
0	—	—	—	133	141	118	—	664	219	255.0
25	63	—	—	84	66	192	154	454	139	164.6
75	76	—	—	65	103	—	66	198	83	98.5
12	—	—	31	41	—	66	45	139	78	66.7
175	—	—	—	41	60	46	66	—	61	54.8
225	—	35	—	34	56	41	48	68	59	46.3
AVE.	—	—	—	66.3	85.2	92.6	75.8	304.6	106.5	112.0
	WASHED									
0	40	40	91	133	136	71	128	492	98	136.6
25	37	23	64	58	85	80	62	262	83	83.8
75	64	34	46	59	80	—	50	77	60	58.8
125	58	43	w	58	55	46	36	59	45	47.3
175	47	35	—	18	46	31	45	—	62	40.6
225	50	31	—	36	58	32	31	44	43	40.6
AVE.	49.3	34.3	56.8	60.3	76.7	52.0	58.7	186.8	65.2	69.8

Note: Lead concentrations in ppm; wet or dry weight not specified.

Source: Motto *et al.*, 1970

SUMMARY

- Exposure to lead may be direct or indirect. There is a continuous transfer of lead between air, water and soil. Knowledge of the transport and eventual fate of lead in the environment is required to assess indirect exposure to lead.
- Ambient air lead levels in Ontario have decreased significantly, particularly during the 1980's when the mean monitored levels in urban areas dropped from 0.3 to 0.01 g/m³. Levels in rural areas are close to the analytical detection limit of 0.01. The only recent exceedances of the 24-hour air standard for lead have occurred in the vicinity of secondary lead smelters. Soils and sediments are the primary sinks for lead deposition through the atmosphere. Dry deposition is significant in the areas surrounding secondary lead smelters, where violations of the point of impingement guideline (0.1 g/m²/30 days) have been frequent.
- Elevated levels of lead in Ontario drinking water are primarily due to a combination of plumbosolvent water and lead plumbing or copper plumbing with lead solder. The Drinking Water Surveillance Program has reported flushed samples to be steadily declining from 3.0-4.0 µg/L (mean median) in 1985-1987 to 0.47 µg/L (mean average) in 1991.
- In Ontario, the major loadings of lead into the Great Lakes appear to be due to atmospheric deposition. Most of the lead is trapped in sediment. It appears that the lead concentrations in the Great Lakes, measured either as dissolved lead or suspended particulate, have declined since the 1970's. Lead in sediment also appear to have declined. Factors affecting sediments, their bulk movement or capacity for lead, have the greatest effect on water lead concentrations and hence, the lead levels in aquatic biota. However, elevated lead levels in both water and sediment have been measured near industrial point sources.
- Fish can be used indicators of the lead burden in the aquatic environment. Elevated lead levels have been found in samples of fish taken from the immediate vicinity of an industrial point source.
- The major source of soil lead is atmospheric deposition. Atmospheric lead enters soil primarily as the insoluble lead sulphate. Hence, lead tends to be retained strongly in soil, with little tendency to migrate into groundwater. Because lead is ubiquitous in nature, even soils in remote rural areas will have some quantity of lead. Levels in rural areas will be affected by organic content as well as by agricultural soil management practices. Generally, rural levels in Ontario are below 50 ppm and urban areas about an order of magnitude higher. The Ministry of the Environment has set 500 ppm as the upper limit of normal for soil lead in urban areas. Considerably higher levels may occur near motorways, houses with peeling paint or industrial point sources.

Terrestrial animals, both vertebrate and invertebrate, may have elevated body lead burdens as a result of environmental exposure, but do not appear to present an important route of human exposure. Terrestrial plants, however, can absorb lead, although the extent of uptake and the site of lead accumulation are the topic of debate. Generally, plants will reflect soil lead levels and lead accumulates in the root system. Consumption of vegetables from urban gardens with elevated soil lead may present a significant source of human exposure.

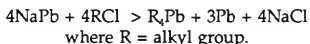
APPENDIX E

ORGANOLEAD COMPOUNDS

APPENDIX E ORGANOLEAD COMPOUNDS

Environmentally, the most important organic compounds of lead are tetraethyl lead (TEL) and tetramethyl lead (TML) forms, because of their use as motor antiknock agents in gasoline. They are being phased out in favour of less polluting substitutes.

Commercial production is achieved via alkylation of a sodium lead alloy according to the reaction:



The properties of organolead compounds are substantially different from inorganic lead compounds. Organoleads are colourless, apolar, moderately volatile liquids. They are insoluble in water, but highly soluble in numerous organic solvents. Unlike inorganic lead compounds, they are lipid soluble, a factor which contributes to their greater toxicity (Environment Canada, 1985).

The manufacture, transport, and handling of gasoline are the major sources of organolead vapours. However, organoleads are photoreactive and will not persist in the atmosphere. Tetraethyl lead (TEL) and tetramethyl lead (TML) exist primarily in the vapour phase. They are photoreactive and will decompose to trialkyl and dialkyl compounds. Trialkyl lead is expected to occur in the vapour form and dialkyl lead primarily in the particulate form. The latter two compounds are highly soluble so that removal from the atmosphere via scavenging precipitation is likely. Due to the particulate nature of dialkyl lead, dry deposition is also a possible removal process (ATSDR, 1988). Organoleads have been found to contribute less than 10% of the total lead present in the atmosphere (ATSDR, 1988).

TEL and TML are insoluble in water and tend to be adsorbed by suspended particulate matter and sediment. This may extend their persistence in the environment. Both compounds may undergo hydrolysis or photolysis. Photolysis of TML produces TEL which is even more persistent in the environment. Tetraalkyl lead compounds degrade to trialkyl lead, dialkyl lead and eventually inorganic lead (ATSDR, 1988).

Limited information indicates that TML and TEL are converted to water soluble lead compounds. These degradation products may leach into water sources (ATSDR, 1988).

Bioaccumulation of organoleads does occur, primarily in fish. However, with the phase-out of lead in gasoline, high levels in aquatic biota are not expected (Environment Canada, 1991).

APPENDIX F

AGE CLASSES, BODY WEIGHTS AND INTAKES

APPENDIX F AGE CLASSES, BODY WEIGHTS AND INTAKES

The following values were used in estimating multi-media exposure to lead for members of the Ontario general population. The sources for these quantities are indicated as footnotes to the table.

TABLE F-1 VALUES FOR AGE-SPECIFIC INTAKES AND BODY WEIGHTS USED TO ESTIMATE ONTARIO GENERAL POPULATION EXPOSURE TO LEAD

Age (yr)	Body Weight (kg)	Air Intake (m ³ /d)	Water Intake ¹ (L/d)	Soil Intake ² (mg/d)
0 - 0.5	7 ³	2 ⁴	0.1 ⁵	35
0.5 - 4	13 ³	5 ⁶	0.8 ⁷	80
5 - 19	42 ¹⁰	18 ^{2,9}	1.1 ¹⁰	35
20 - 70	70 ²	20 ²	1.5 ¹¹	20

- ¹ - "Tap Water Consumption in Canada" (HWC, 1983)
- ² - "Reference Values for Canadian Populations" (HWC, Draft)
- ³ - Nutrition Canada Survey (HWC, 1977)
- ⁴ - Midpoint between ICRP (1975) values of 0.8 m³ for newborn and 3.8 m³ for 1-year-old infant
- ⁵ - Arbitrary
- ⁶ - Linear regression between 3.8 m³ @ 1 yr (ICRP, 1975) and 9.25 m³ @ 7 yr (HWC, Draft); predicted inhaled volume at midpoint of 0.5 - 4 yr age class equals 4.9 m³ per day
- ⁷ - Weighted mean of <3 yr and 3-5 yr categories
- ⁸ - Canada Health Survey (Statistics Canada/NWH, 1981)
- ⁹ - Mean for both sexes, ages 7-19
- ¹⁰ - Mean, ages 6-17
- ¹¹ - Mean, adults 18 and over

The age classes are similar to those used in previous multi-media exposure assessments. The above intakes and body weights have been rounded off, since the actual values for these quantities are likely to be quite variable between individuals (for example, minute volumes vary as a function of activity, weight, etc.)

APPENDIX G

**ESTIMATED DAILY INTAKES OF LEAD
IN FOOD IN ONTARIO**

APPENDIX G ESTIMATED DAILY INTAKES OF LEAD IN FOOD IN ONTARIO

The estimated daily lead intake from food is calculated by multiplying the consumption rate for an individual food category (Table 4.2, G-1, G-2, G-3) by the average concentration of lead in that food group and summing the subtotals. In this exposure analysis, the food lead concentrations from the Duplicate Diet Study were chosen over the incomplete Ontario data, as the former are a complete diet set and thereby more representative of the actual combination of levels experienced by an individual (Dabeka *et al.*, 1987). Also, the study used a highly sensitive detection methodology and the data are internally consistent across food groups, all being analyzed similarly. Although the food categories used in this study do not exactly correspond to those of the Nutrition Canada Survey, they are sufficiently similar to make rough estimates of intake. The foods in the "Miscellaneous" and "Primarily sugar" categories do vary significantly between the two studies so a value of 0.117 $\mu\text{g/g}$ was assumed for the "Primarily sugar" group; this is the mean value of the Dabeka "Miscellaneous" category. The "Miscellaneous" and "Oil and Fats" groups were assigned levels of 0.100 $\mu\text{g/g}$, a conservative value lying between the mean and median values of Dabeka's grouping.

The results of the analysis are shown in Tables G-4, G-5, and G-6. The estimated total daily intakes increase from 36.5 $\mu\text{g/day}$ for children 1-4 years old to a maximum of 64.1 $\mu\text{g/day}$ for teenage males. Women tended to have lower total intakes, largely attributable to lower overall consumption rates. The calculated average intake for teenagers is 59.2 $\mu\text{g/day}$ or 1.0 $\mu\text{g/kg/day}$ based on an average body weight of 57.2 kg. For adults, this figure is 45.7 $\mu\text{g/day}$ or 0.65 $\mu\text{g/kg/day}$ based on a body weight of 70 kg. Pregnant women consume an estimated 52.4 $\mu\text{g/day}$ (tables G-5 and G-6). On a per unit body weight basis, intakes for children in the 1-4 and 5-11 year groups are considerably larger than for the teenage/adult groups.

Examination of figures from a similar exercise conducted by Health and Welfare Canada using similar databases reveal comparable results for the Canadian population as a whole, particularly for the 1-4 and 5-11 year old children (Chapter 4 Section 4.3.2.2). In this analysis, adults 20 years or older were considered as a single group. This, however, does make comparison with the adult groupings in Table 4.2 less direct.

TABLE G-1 AVERAGE DAILY FOOD CONSUMPTION RATES AMONG ONTARIO SUBPOPULATIONS¹

FOOD CATEGORY	CONSUMPTION (G/DAY)					
	Child (1-4)	Child (5-11)	Male (12-19)	Female (12-19)	Adult Male ² (20+)	Adult Female ² (20+)
Milk and Dairy Products	626	696	813	563	355	296
Meat, Fish, Poultry	62	88	173	73	190	106
	82	108	208	118	225	141
Eggs	20	20	30	30	30	30
Cereal Products	144	245	368	239	248	185
Fruits, Fruit Products	262	305	263	287	222	234
Vegetables (not potatoes)	49	71	104	95	130	113
Potatoes	61	99	172	74	137	77
Oils and Fats	11	19	36	17	24	17
Foods - Primarily Sugar	26	39	53	29	52	30
Nuts and Legumes	4	8	26	21	9	70
Miscellaneous	109	118	118	112	111	70
TOTAL	1411	1745	2049	1565	1513	1173
SAMPLE SIZE	178	326	203	202	-	-

¹ Based on average food consumption figures from Nutrition Canada Survey (NCS, 1977).

TABLE G-2 INFANT FOOD CATEGORIES AND LEAD LEVELS²

CATEGORY	NO. OF SAMPLES	MEAN (ng/g)	MEDIAN	RANGE
1. INFANT FORMULAS				
A. Concentrated liquid formula	30	21.0	15.9	1.2 - 67
- lead free cans	8	3.5	2.4	1.2 -9.8
- lead soldered cans	22	27.4	22.2	5.8 - 67
B. Ready-to-use formula (canned)	15	37.3	26.0	1.1 -122
- glass-bottled (fed in hospitals)	20			
- lead free cans	3	2.5	2.2	0.1 -4.5
- lead soldered cans	12	1.7	1.6	1.5 -2.0
		46.2	30.1	1.1 -111
C. Concentrated powder formula	31			
- collected in 1978	25	73.7	45.	3.7 -532
- collected in 1985	6	88.7	48.	9 - 532
		11.5	6.6	3.7 - 19
2. Meat and meat dinners	30	19.3	12.3	0.3-56.7
3. Vegetables and vegetable combinations	24	8.4	5.6	3.7 - 34
4. Fruits and desserts	24	14.9	8.2	2.3 - 65
5. Juices and drinks for infants	24	9.6	7.4	3.5-26.5
6. Infant cereals (dry)	39	32.8	25.8	7.6 -121
7. Evaporated milk	8	72.4	71.9	27 - 106
8. Cow milk (market)	68	1.12	1.19	.01 -2.5
9. Human milk	210	1.04	0.57	<.05-15.8
10. Table foods	164	31.9 ²	23.4 ²	.62 -254
11. Water	18	8.8	2.0	0.25 -71

¹ Source: Dabeka and McKenzie, 1987² Mean overall samples in 8 food categories; Source: Dabeka *et al.*, 1987

TABLE G-3 LEVELS OF LEAD IN FRUITS, JUICES, DESSERTS, MEATS, VEGETABLES AND CEREALS

FOOD	NO. OF SAMPLES	LEAD ($\mu\text{g/g}$)	
		MEAN	RANGE
FRUIT JUICES			
- mixed juice	6	10.9	6.4 - 16
- apple juice	6	7.7	5.3 - 10.5
- orange juice	6	4.8	3.5 - 6.9
- apple cherry juice	6	15.7	6.6 - 28.5
FRUITS AND DESSERTS			
- bartlett pears	3	16.0	13 - 19
- peaches	6	8.7	5 - 17
- vanilla custard	3	4.0	3 - 6
- orange delight	3	4.0	3 - 5
- apricots	3	37.2	23 - 47
- apple sauce	3	32.4	14 - 65
- bananas	3	8.97	7.3 - 12
VEGETABLES			
- peas	6	6.8	5 - 13
- green beans	3	6.6	5 - 8
- squash	3	5.3	5 - 6
- carrots	6	6.3	4 - 10
- garden vegetables	3	5.9	5.1 - 7.1
- sweet potato	3	23.1	16 - 34
MEATS			
- turkey broth	6	21.0	40.9 - 41.3
- veal	8	20.9	3.2 - 57
- beef	9	13.6	3.0 - 33.2
- chicken broth	6	22.2	9.6 - 35.3
CEREALS (DRY)			
- oatmeal	7	17.5	7.6 - 32
- rice	9	41.9	14.3 - 76
- barley	5	17.3	11.6 - 23
- mixed	14	36.8	11.7 - 121
- biscuits and other	4	42.7	28.8 - 69.5

TABLE G-4 ESTIMATED DAILY LEAD INTAKE FROM FOODS IN ONTARIO SUBPOPULATIONS

FOOD CATEGORY	LEAD CONC. (µg/g)	ESTIMATED INTAKE (µg/day)					
		Child (1-4)	Child (5-11)	Male (12-19)	Female (12-19)	Adult Male (20+)	Adult Female (20+)
Milk and Dairy Products	0.009	5.63	6.26	7.32	5.07	3.20	2.66
Meats, Poultry and Fish	0.043	2.67	3.78	7.44	3.14	8.17	4.56
Eggs	0.009 (See Dairy)	0.18	0.18	0.32	0.32	0.32	0.32
Cereal Products	0.034	4.32	8.33	12.51	8.13	8.43	6.29
Fruits	0.018	4.72	5.49	4.73	5.17	4.00	4.21
Vegetables (not potatoes)	0.049	2.40	3.48	5.10	4.66	6.37	5.54
Potatoes	0.022	1.34	2.18	3.78	1.63	3.01	1.69
Oils and Fats	0.100	1.1	1.9	3.6	1.7	2.4	1.7
Foods - Primarily Sugar	.117	3.04	4.56	6.20	3.39	6.08	3.51
Nuts and Legumes	0.049 (See Veg's)	0.20	0.39	1.27	1.03	.044	0.49
Miscellaneous	0.100	10.4	11.8	11.8	11.2	11.1	7.0
TOTAL DAILY INTAKE (µg/day)	-	36.5	48.4	64.1	45.4	53.5	37.9
INTAKE PER UNIT BODY WEIGHT (µg/kg/day)		2.52	1.84	1.07	0.84	0.70	0.61

TABLE G-5 ESTIMATED MEAN DAILY INTAKE OF LEAD IN FOOD BY PREGNANT WOMEN IN ONTARIO

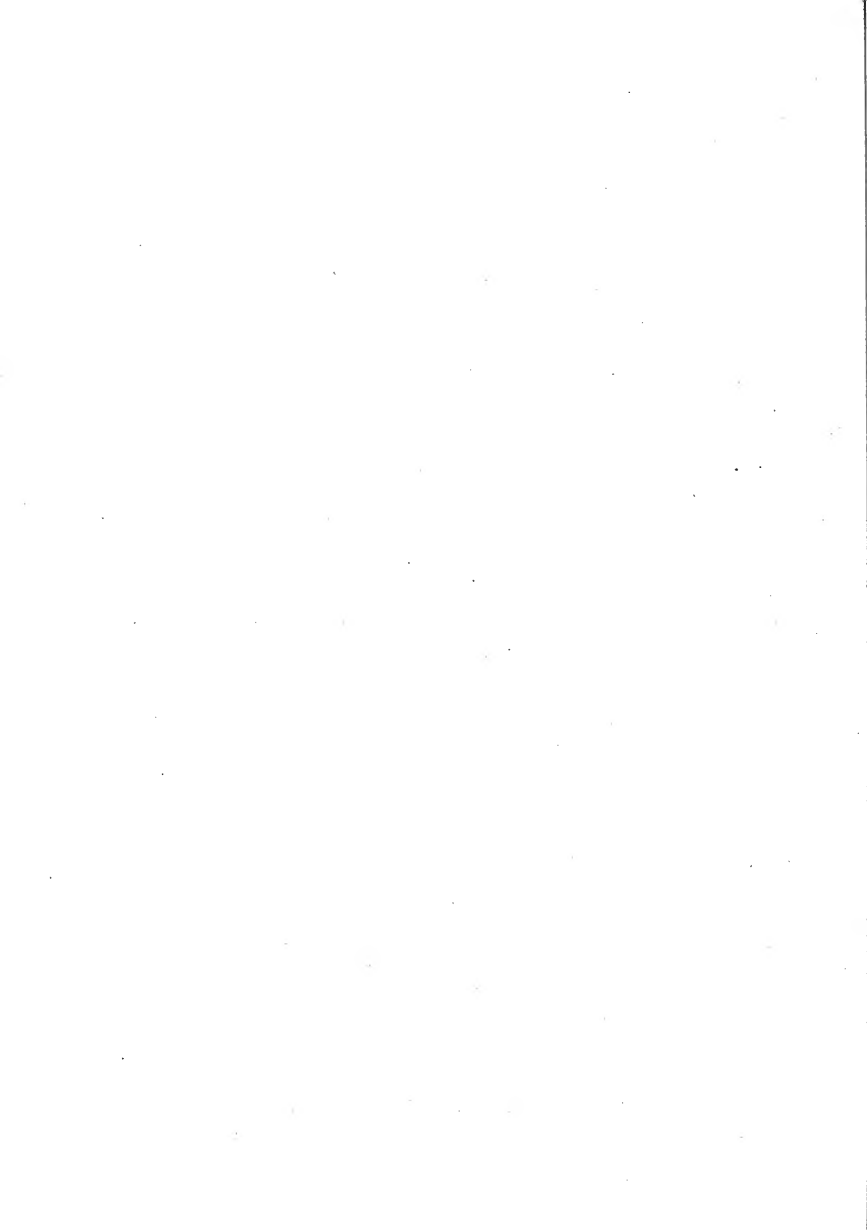
FOOD CATEGORY	FOOD INTAKE (g/day)	ESTIMATED LEAD INTAKE (µg/day)
Milk and Dairy Products	629	5.66
Meats, Poultry and Fish	120	5.16
Eggs	35	0.32
Cereal Products	185	6.29
Fruits	317	5.71
Vegetables (not potatoes)	153	7.50
Potatoes	90	1.98
Oils and Fats	19	1.90
Food - primarily sugar	42	4.91
Nuts and Legumes	17	0.83
Miscellaneous	121	12.10
TOTAL DAILY INTAKE	1728	52.36

TABLE G-6 ESTIMATED MEAN DAILY INTAKE OF LEAD IN FOOD BY PREGNANT WOMEN AND WOMEN OF CHILD-BEARING AGE IN ONTARIO

FOOD CATEGORY	FOOD INTAKE (µg/day)		EST. LEAD INTAKE (µg/day)	
	Pregnant Women	Women (20-39)	Pregnant Women	Women (30-39)
Milk and Dairy Products	629	322	5.66	2.90
Meats, Poultry and Fish	120	126	5.16	5.42
Eggs	30	30	0.27	0.27
Cereal Products	185	214	6.29	7.28
Fruits	317	236	5.71	4.25
Vegetables (not potatoes)	153	106	7.50	5.19
Potatoes	90	83	1.98	1.83
Oils and Fats	19	20	1.90	2.0
Food-primarily sugar	42	32	4.91	3.7
Nuts and Legumes	17	7	0.83	0.34
Miscellaneous	121	77	12.10	7.70
TOTAL DAILY INTAKE	1723	1303	52.31	40.88

APPENDIX H

**DERIVATION OF
THE CENTERS FOR DISEASE CONTROL
RECOMMENDED BLOOD LEAD INTERVENTION LEVELS**



APPENDIX H DERIVATION OF THE CENTERS FOR DISEASE CONTROL
RECOMMENDED BLOOD LEAD INTERVENTION LEVELS

TABLE H-1 INTERPRETATION OF BLOOD LEAD TEST RESULTS AND FOLLOW-UP
ACTIVITIES

CLASS	BLOOD LEAD CONCENTRATION ($\mu\text{g}/\text{dL}$)	COMMENT
I	< 9	A child in Class I is not considered to be lead-poisoned.
IIA	10-14	Many children (or a large proportion of children) with PbB in this range should trigger community-wide childhood lead poisoning prevention activities. Children in this range may need to be rescreened more frequently.
IIB	15-19	A child in Class IIB should receive nutritional and educational interventions and more frequent screening. If the PbB persists in this range, environmental investigation and intervention should be done .
III	20-44	A child in Class III should receive environmental evaluation and remediation and a medical evaluation. Such a child may need pharmacologic treatment of lead poisoning.
IV	45-69	A child in Class IV will need both medical and environmental interventions, including chelation therapy.
V	>70	A child with Class V lead poisoning is a medical emergency. Medical and environmental management must begin immediately.

Source: CDC, October 1991

The single all-purpose definition of childhood lead poisoning has been replaced with a multi-tier approach, (Table H-1). Community prevention activities should be triggered by blood lead levels greater than 10 $\mu\text{g}/\text{dL}$. Medical evaluation and environmental investigation and remediation should be done for all children with PbB greater than 20 $\mu\text{g}/\text{dL}$. All children with PbB greater than 15 $\mu\text{g}/\text{dL}$ should receive individual case management, including nutritional and educational interventions and more frequent screening. Furthermore,

depending on the availability of resources, environmental investigation (including a home inspection) and remediation should be done for children with PbB ranging from 15 to 19 $\mu\text{g}/\text{dL}$, if such levels persist. The highest priority should continue to be the children with the highest PbB.

APPENDIX I

REGULATORY SUMMARY

APPENDIX I REGULATORY SUMMARY

This chapter is an assembly of current international guidelines, objectives, standards and limiting concentrations for lead in the various media: air, water (drinking water, ambient water), soil, food, and consumer products. The status of the regulations is reported to indicate of whether revisions are eminent. Where available, the basis for the limits has been included.

TABLE I-1 INTERNATIONAL LEAD REGULATIONS: AIR

AGENCY	LIMIT	REGULATORY STATUS	BASIS	DESCRIPTION
CANADA				
Federal Regulations: Ambient air	2 µg/m³	S		30 day geometric mean
	5 µg/m³	S		24 hour maximum
Secondary lead smelter (1989)	0.046 g/m³	S	environmental health and human health	blast, cupolas or reverberatory furnaces
	0.023 g/m³	S	"	holding or kettle furnaces
Manitoba	5 µg/m³	G	-	24 hour exposure period
Quebec	0.2 µg/m³	G	-	annual exposure period
Newfoundland	5 µg/m³	G	-	24 hour exposure period
	2 µg/m³	G	-	30 day exposure period
Montreal Urban Community	10 µg/m³	G	-	1 hour exposure period
	5 µg/m³	G	-	8 hour exposure period
Ontario: Ambient air (1972)	5 µg/m³	G	environmental health, human health	24 hour maximum
	3 µg/m³	G	"	30 day arithmetic mean
	2 µg/m³	G	"	30 day geometric mean
	10 µg/m³	S	"	1/2 hour point of impingement average
Dustfall (1974)	0.1 g/m²/30 days	G	human health, limits soil contamination rates, prevents a more than 3-fold increase above the normal urban level of lead in dustfall and limits the lead content of total dustfall to less than 2% by weight	point of impingement guideline

AGENCY	LIMIT	REGULATORY STATUS	BASIS	DESCRIPTION
Alberta: Secondary lead smelter	0.029 g/m ³ 0.014 g/m ³	S S	- -	blast furnace, cupolas, reverberatory furnaces holding furnaces, pot furnaces, lead oxide production units or any other melting or refining operations
British Columbia Ambient air control objectives (1979) Ambient air quality guidelines	1-2.5 µg/m ³ 4 µg/m ³ 4 µg/m ³ 6 µg/m ³	O G G G	ecological, health, technological and economic considerations " - -	- 24 hour maximum desirable level 24 hour maximum acceptable level 24 hour maximum tolerable level
Pollution control objectives	2 µg/m ³ 7 mg/m ³ 0.5 kg/tonne 7 mg/m ³ 0.9 lb/tonne	G O O O O	ecological, health, technological and economic considerations " " " "	1 year geometric mean chlor-alkali and sodium chlorate industries lead smelting and refining all food-processing and miscellaneous industries secondary lead smelting

AGENCY	LIMIT	REGULATORY STATUS	BASIS	DESCRIPTION
UNITED STATES				
USA				
OAQPS: national ambient air quality standards	1.5 µg/m³	S	human health effects	maximum arithmetic mean over calendar quarter
OSHA: permissible exposure limit	50 µg/m³	S	"	8-hour TWA
action level	30 µg/m³	S	"	8-hour
ACGIH: threshold limit value (TWA)	0.15 mg/m³	G	"	inorganic lead, dust and fumes
NIOSH: recommended exposure level	<0.1 mg/m³	G	"	10-hour TWA
USEPA: OAR				
Lead-acid battery manufacturing plants	>0.40 mg/m³	S	-	grid-casting facilities
	>1.0 mg/m³	S	-	paste-mixing facility
	>1.0 mg/m³	S	-	three-process operation facility
	>5.0 mg/kg	S	-	lead oxide manufacturing
	>4.5 mg/m³	S	-	lead reclamation
	>1.0 mg/m³	S	-	lead emitting operation

AGENCY	LIMIT	REGULATORY STATUS	BASIS	DESCRIPTION
Acceptable Ambient Air Concentrations				
California	1.5 $\mu\text{g}/\text{m}^3$	S	-	30 day arithmetic mean
Connecticut	3.0 $\mu\text{g}/\text{m}^3$	G	-	8-hour average
Kansas	0.357 $\mu\text{g}/\text{m}^3$	G	-	1-year average
Massachusetts	0.140 $\mu\text{g}/\text{m}^3$	G	-	24-hour average
	0.070 $\mu\text{g}/\text{m}^3$	G	-	annual average
North Dakota	0.0015 mg/m^3	G	-	8-hour average
Nevada	0.004 mg/m^3	G	-	8-hour average
Pennsylvania/Philadelphia	1.50 $\mu\text{g}/\text{m}^3$	G	-	annual and 1-year average
Virginia	2.50 $\mu\text{g}/\text{m}^3$	G	-	24-hour average
Vermont	1.5 $\mu\text{g}/\text{m}^3$	G	-	3-month average
Ambient Air Emissions Limitations				
Kentucky	0.1 $\mu\text{g}/\text{m}^3$	G	-	3-month average
Montana	0.1 $\mu\text{g}/\text{m}^3$	G	-	24-hour average
Permit required to construct & operate an air contamination source project if yearly emissions exceed:				
Arizona, Connecticut, Missouri, New York, Virginia	0.6 ton	G	-	-
Sources exempt from air monitoring requirements if net emissions increase is less than:				
Delaware, Louisiana, Oregon, Wisconsin	<0.1 $\mu\text{g}/\text{m}^3$	G	-	24-hour average

AGENCY	LIMIT	REGULATORY STATUS	BASIS	DESCRIPTION
EEC	2 $\mu\text{g}/\text{m}^3$	-	-	maximum limit
	150 $\mu\text{g}/\text{m}^3$	-	-	time weighted average
Germany	0.1 mg/m^3	-	-	8 hour TWA
WHO	30-60 $\mu\text{g}/\text{m}^3$	-	-	maximum limit

Level A category (objective for new and proposed discharges and within the limits of the best practicable technology, to existing discharges by planned staged improvements)

S = STANDARD

O = OBJECTIVE

G = GUIDELINE TWA = TIME WEIGHTED AVERAGE

TABLE 1-2 INTERNATIONAL LEAD REGULATIONS: DRINKING WATER

AGENCY	LIMIT	REGULATORY STATUS	BASIS	DESCRIPTION
CANADA	0.01 mg/L	G	Human health effects (flushed sample)	Maximum allowable concentration
Ontario	0.01 mg/L	G	"	Adopts the Canadian Drinking Water Guidelines, enforceable under the Ontario Water Resources Act
USA: USEPA ODW	0.015 mg/L	S	-	Action level
	0 mg/L	-	based on a standing sample	Regulated under the Safe Drinking Water Act, 1986
	0.02 mg/L	PG	Based on a long-term health advisory	Maximum contaminant level goal
Maine	20 µg/L	G	-	Recommended maximum contaminant level
Alabama	0.02 mg/L	-	-	MAC
Iowa	0.05 mg/L	-	-	MAC
Texas	0.05 mg/L	-	-	MAC
Illinois	50 µg/L	PS	-	-
Indiana	50 µg/L	S	-	-
Minnesota	50 µg/L	S	-	-
	20 µg/L	PS	-	-
New York	50 µg/L	S	Human health effects	Ambient water quality standards, regulations for drinking water supplies
	25 µg/L	S	"	Ground water quality standards, regulations for ground water
Kentucky	0.05 mg/L	S	-	Domestic water supply source criteria
	0.05 mg/L	S	-	Maximum ground water contaminant level

AGENCY	LIMIT	REGULATORY STATUS	BASIS	DESCRIPTION
Nevada	0.05 mg/L	G	-	Interim primary drinking water standards
	0.05 mg/L	G	-	Municipal or domestic water supply
Florida	0.050 mg/L	S	Human health effects	Maximum contaminant level
Germany	0.04 mg/L	S	-	Maximum permissible concentration
WHO	0.05 mg/L	-	Human health effects	-
The former USSR	0.100 mg/L	S	Human health effects	Maximum permissible concentration

ODW: Office of Drinking Water

OWRS: Office of Water Regulations and Standards

MAC: Maximum Contaminant Concentration

S: Standard

G: Guideline

P: Proposed

O: Objective

TABLE I-3 INTERNATIONAL LEAD REGULATIONS: WATER QUALITY

AGENCY	SUBSTANCE NOTES	WATER USE	LEVEL	TYPE*	RATIONALE	REGULATORY STATUS
CCME	All soils	Irrigation	200 µg/L	-	soil accumulation	P
		Ambient	1 µg/L	A	USEPA chronic toxicity	S
		Ambient	2 µg/L	A	USEPA chronic toxicity	S
		Ambient	4 µg/L	A	USEPA chronic toxicity	S
		Ambient	7 µg/L	A	USEPA chronic toxicity	S
		Livestock	100 µg/L	-	livestock toxicity	S
PROVINCE OF ONTARIO	Unfiltered water	Ambient	5 µg/L (<20 mg/L CaCO ₃)	A	toxicity to rainbow trout	G
	"	"	10 µg/L (20-40 mg/L CaCO ₃)	A	"	G
	"	"	20 µg/L (40-80 mg/L as CaCO ₃)	A	"	G
	"	"	25 µg/L (>80 mg/L as CaCO ₃)	A	"	G
	-	Ambient	1 µg/L (0-30 mg/L as CaCO ₃)	-	"	PG
	-	Ambient	3 µg/L (30-80 µg/L as CaCO ₃)	-	"	PO
	-	Ambient	5 µg/L (>80 µg/L as CaCO ₃)	-	"	PO
	-	Livestock	100 µg/L	-	livestock guideline	G
	-	Irrigation	5000 µg/L	-	Irrigation guideline	G
	-					
PROVINCE OF BRITISH COLUMBIA	effluent discharge to marine and fresh waters from metal finishing plants and industries discharging heavy metals	-	0.05 - 0.2 mg/L	-	-	O

AGENCY	SUBSTANCE NOTES	WATER USE	LEVEL	TYPE*	RATIONALE	REGULATORY STATUS
IJC GREAT LAKES WATER QUALITY AGREEMENT	Erie & Michigan	Ambient	4.0 µg/L	A	chronic effects	R
	Lake Huron	Ambient	3.0 µg/L	A	chronic effects	
	Lake Ontario	Ambient	5.0 µg/L	A	neurotoxic effects on trout	
	Lake Superior	Ambient	2.0 µg/L	A	chronic effects	
USEPA: OWRS	1 h average	Ambient	82 µg/L	A	chronic toxicity to freshwater life	G
	4 d average	Ambient	3.2 µg/L at 100 mg/L	A	acute toxicity to freshwater life	G
	1 h average	Ambient	140 µg/L	A	chronic toxicity to marine life	G
	4 day average	Ambient	5.6 µg/L	A	acute toxicity to marine life	G
STATE OF ARIZONA	-	Ambient	50 µg/L	H	human toxicity	G
	-	-	> 5 ton/year	-	permit required for operation emitting amounts greater than limit	C
STATE OF CALIFORNIA	6-month median	-	2 µg/L	-	protection of marine and aquatic life	O
	daily maximum	-	8 µg/L	-		O
	instantaneous maximum	-	20 µg/L	-		O
STATE OF ILLINOIS	Total	Ambient	100 µg/L	-	general water use	S
STATE OF INDIANA	Acid soluble	Ambient	exp. (1.2731/in hardness/4.705)	A	protect aquatic life/chronic effects	P
	Acid soluble	Ambient	exp. (1.273/in hardness/-1.460)	A	prevent acute effects	P
IOWA (surface water quality criteria)	-	-	0.1 mg/L	-	protected for wildlife, fish, aquatic and semi aquatic life	C
	-	-	0.05 mg/L	-	protected as a raw water source of potable water supply	C

AGENCY	SUBSTANCE NOTES	WATER USE	LEVEL	TYPE	RATIONALE	REGULATORY STATUS
STATE OF MICHIGAN: DNR	-	NPDES - National Point Discharge Elimination System	exp.(1.75/in hardness/ - 7.00)	A	aquatic chronic value	S
NEVADA (water quality criteria)	-	-	< 5.0 mg/L	-	irrigation	C
	-	-	< 0.1 mg/L	-	watering of livestock	C
	-	-	< 0.1 mg/L	-	propagation of wildlife	C
NEW MEXICO	-	-	0.05 mg/L	-	groundwater standards	R
NEW YORK STATE: DEC	Acid soluble	Fish life	exp. (1.266/in hardness - 1.416)	A	USEPA	S
	Acid soluble	Fish	exp. (1.266/in hardness - 4.661)	A	USEPA	S
STATE OF OHIO	-	Ambient	30.00 µg/L	A	aquatic life/general uses	S
STATE OF PENNSYLVANIA	-	All	exp (1.266/in hardness - 4.661)	A	criteria continuous concentrations	S
	-	All	exp. (1.266/in hardness - 1.416	-	criteria maximum concentrations	S
UTAH	-	-	0.05 mg/L	-	groundwater standards	S
WISCONSIN	-	public health, ground water quality standards	50 µg/L	-	enforcement standard	S
			5 µg/L		preventative action limit	S

*Methodology for Guideline Development H: Human Health A: Aquatic Life E: Ecosystem

S = STANDARD; P = PROPOSED; C = CRITERIA; O = OBJECTIVE; G = GUIDELINE

OWRS = OFFICE OF WATER REGULATIONS AND STANDARDS DNR = DEPARTMENT OF NATURAL RESOURCES
 DEC = DEPARTMENT OF ENVIRONMENTAL CONSERVATION IJC = INTERNATIONAL JOINT COMMISSION

TABLE I-4 INTERNATIONAL LEAD REGULATIONS: SOIL

AGENCY	LIMIT	REGULATORY STATUS	BASIS	DESCRIPTION
ONTARIO Decommissioning Guidelines				
medium & fine textured soils	500 µg/g	G	consideration of human health, animal health and phytotoxicology	agricultural, residential, parkland
	1000 µg/g	G	"	commercial, industrial
course textured soils	375 µg/g	G	"	agricultural, residential, parkland
	750 µg/g	G	"	commercial, industrial
Soil near secondary lead smelters	500 µg/g	G	-	same as decommissioning guidelines for medium & fine textured soils
	1000 µg/g	G	-	
Upper Limits of Normal				
	150 µg/g	G	statistically-bound upper limit of background contaminant levels in urban and rural soils (toxic effects not expected at levels below ULN's)	rural
	500 µg/g	G		urban
Excavated Material Guidelines	0.05 mg/L	G	based on leachate quality criteria	
Open Water Disposal Guideline	50 µg/g	G	protection of existing water uses	Intended to be superseded by the Provincial Sediment Quality Guidelines
Dredged Material and Backfill Material Guidelines	60 ppm (unrestricted land use)	G	protection of environmental quality by managing dredged material consistent with physical/chemical characteristics	must meet open water disposal guidelines (50 µg/g)
	500 ppm (restricted land use)	G	biological effects-based	
Sediment Quality Guidelines	31 µg/g	PG	based on the lowest-observed adverse effect level	protection for all water uses

AGENCY	LIMIT	REGULATORY STATUS	BASIS	DESCRIPTION
Sewage Sludge Utilization Guidelines	60 µg/g 90 kg/ha	G G	promote environmentally safe and agriculturally beneficial use of a waste that would otherwise require incineration or landfill disposal	maximum permissible content in soil maximum permissible addition to soil
Compost Guidelines	150 ppm	G	-	upper limits of normal
Snow	0.07 mg/L melted snow	G	-	
Vegetation (upper limits of normal)	urban			
	60 µg/g	G	-	tree foliage (live, unwashed)
	200 µg/g	G	-	moss bag
	rural			
	30 µg/g	G	-	tree foliage (live, unwashed)
Ontario Typical Range	20 µg/g	G	-	grass (live, unwashed)
	35 µg/g	G	-	moss bag
	urban - 98 µg/g rural - 45 µg/g	- -	represents the expected range of concentrations for lead in surface soil taken from areas removed from known point sources	an action level, OTR98, has been defined which represents 98% of the data in the OTR distribution
USA				
Minnesota	soil 300 ppm	S	protective of human and environmental health	bare soil standard
	dust 500 ppm	S	"	interior or exterior residential dust
	80 µg/ft ²	S	"	hard-surfaced floor
	300 µg/ft ²	S	"	windowsill
	500 µg/ft ²	S	"	window well
Massachusetts	300 ppm	S	"	cleanup number based on exposure to the sensitive receptor (intensity and frequency)

P = PROPOSED O = OBJECTIVE G = GUIDELINE S = STANDARD

TABLE I-5 SOIL CLEANUP LEVELS AT VARIOUS SUPERFUND SITES

LOCATION (STATE)	CLEANUP NUMBER (mg/kg)	NUMBER BASIS
Maine	248	human health based level
New Jersey	250	derived from the Interim New Jersey soil action levels
New Jersey	250	New Jersey cleanup guidelines
New Jersey	250	New Jersey Department of Environmental Protection cleanup objectives
New Jersey	250	New Jersey cleanup objectives
New Jersey	250	New Jersey soil action levels
New Jersey	317	New Jersey environmental Cleanup and Responsibility Act
New Jersey	400	New Jersey soil cleanup standard (accepted but not promulgated)
Arizona	700	human health/ Arizona State Department of Health Services
Indiana	980	reference dose and soil ingestion only pathway/commercial
New Jersey	100	New Jersey State soil cleanup criteria
South Carolina	166.5	based on future groundwater levels < groundwater cleanup levels
Michigan	20	human health-cancer risk based level
Michigan	70	based on non-carcinogenic risk calculation
Florida	100	human health risk assessment
New Jersey	100	New Jersey environmental cleanup and responsibility
Indiana	210	reference dose and soil ingestion only/residential
Texas	4200	human health based level
Texas	420000	cleanup numbers: commercial lean use only with no leaching to ground water

TABLE I-6 INTERNATIONAL LEAD REGULATIONS: FOOD

AGENCY	LIMITS	DESCRIPTION	BASIS	REGULATORY STATUS
CANADA (1989)	10 ppm	edible bone meal	human health effects	S
	0.5 ppm	fish protein	"	S
	1.5 ppm	tomato paste and sauce	"	S
	0.5 ppm	whole tomatoes	"	S
	0.2 ppm	fruit juice, beverages when ready-to-serve and water in sealed containers other than mineral water or spring water	"	S
	0.15 ppm	evaporated milk, condensed milk and concentrated infant formula	"	S
WHO	0.08 ppm	infant formula ready-to-serve	"	S
	25 ug/kg/week	provisional weekly intake	"	G

S = STANDARD G = GUIDELINE

TABLE I-7 INTERNATIONAL LEAD REGULATIONS: CONSUMER PRODUCTS

PRODUCT	AGENCY	LIMIT	REGULATORY STATUS	BASIS	DESCRIPTION
PAINT	CANADA (1975)	0.5% of total weight of contained solids including pigments film solids and driers	S	human health effects	regulated under Part I* of the HPA regulation in place particularly for furniture and other articles intended for children but also including pencils and artist' brushes
	USA: CPSC	0.06% of total weight of solids or paint film	S	-	HUD requires testing and elimination of lead-based paint in federally funded housing and housing rehabilitation programs, public housing and Indian Reserves
	EEC	>0.1%	-	-	preparations containing this concentration of alkyl are toxic
		0.05%-0.1%	-	-	preparations containing this concentration of alkyl lead are considered harmful
		≥1.0%	-	-	preparations containing this concentration of other lead compounds are considered harmful
CERAMICS (source: lead-based glaze)	CANADA	7 mg/L	-	human health effects	lead release limit; regulated under Part II* of HPA
	USA (based on leaching solution)	7 ppm	A	"	pottery flatware (<25 cm deep)
		5 ppm	A	"	small holloware (<1.1 L)
		2.5 ppm	A	"	large holloware
		7.0 ppm	A	"	silver-plated holloware (used by adults)
	GERMANY	0.5 ppm	A	"	silver-plated holloware (used by children)
		0.3 mg/L	-	-	maximum permissible concentration

PRODUCT	AGENCY	LIMIT	REGULATORY STATUS	BASIS	DESCRIPTION
GASOLINE	CANADA (1990)	5 mg/L	S	human health effects	unleaded
		26 mg/L (average)	S	"	leaded (for use only in tractors, heavy trucks, and boats)
	USA: USEPA OAR	0.05 gpg	S	"	unleaded
		0.1 gpg	S	"	leaded
KETTLES	CANADA	-	S	human health effects; those intended for household use that release lead into water boiled therin	regulated under Part II* of HPA
SOLDER	ONTARIO (1989)	0.2%	S	human health effects	solders and fluxes having lead content in excess of standard not to be used in potable water systems
CRYSTAL	ONTARIO	7 ppm	S	human health effects	maximum lead release in 4% acetic acid solution

HPA = HAZARDOUS PRODUCTS ACT CPSC = CONSUMER PRODUCT SAFETY COMMISSION HUD = DEPARTMENT OF HOUSING AND URBAN DEVELOPMENT
 OAR = OFFICE OF AIR AND RADIATION gpg = GRAMS PER GALLON A = ACTION LEVELS P = PROPOSED G = GUIDELINE O = OBJECTIVE R = REGULATION
 *No product will be advertised sold or imported into Canada if included in Part I of the HPA **No product will be advertised sold or imported into Canada if included in Part II of the HPA

APPENDIX J

CONVERSION TO SYSTEME INTERNATIONAL (SI) UNITS

APPENDIX J: CONVERSION TO SYSTEME INTERNATIONAL (SI) UNITS

Blood Lead

$$1.0 \mu\text{g/dL} = 0.04826 \mu\text{mol/L} \qquad 1.0 \mu\text{mol/L} = 20.72 \mu\text{g/dL}$$

0 $\mu\text{g/dL}$	= 0 $\mu\text{mol/L}$
5 $\mu\text{g/dL}$	= 0.241 $\mu\text{mol/L}$
10 $\mu\text{g/dL}$	= 0.483 $\mu\text{mol/L}$
15 $\mu\text{g/dL}$	= 0.724 $\mu\text{mol/L}$
20 $\mu\text{g/dL}$	= 0.965 $\mu\text{mol/L}$
25 $\mu\text{g/dL}$	= 1.206 $\mu\text{mol/L}$
30 $\mu\text{g/dL}$	= 1.448 $\mu\text{mol/L}$
35 $\mu\text{g/dL}$	= 1.689 $\mu\text{mol/L}$
40 $\mu\text{g/dL}$	= 1.930 $\mu\text{mol/L}$
45 $\mu\text{g/dL}$	= 2.172 $\mu\text{mol/L}$
50 $\mu\text{g/dL}$	= 2.413 $\mu\text{mol/L}$
55 $\mu\text{g/dL}$	= 2.654 $\mu\text{mol/L}$
60 $\mu\text{g/dL}$	= 2.896 $\mu\text{mol/L}$
65 $\mu\text{g/dL}$	= 3.137 $\mu\text{mol/L}$
70 $\mu\text{g/dL}$	= 3.378 $\mu\text{mol/L}$

Erythrocyte Protoporphyrin

$$1.0 \mu\text{g/dL} = 0.01778 \mu\text{mol/L} \qquad 1.0 \mu\text{mol/L} = 56.25 \mu\text{g/dL}$$

28 $\mu\text{g/dL}$	= 0.498 $\mu\text{mol/L}$
35 $\mu\text{g/dL}$	= 0.622 $\mu\text{mol/L}$
70 $\mu\text{g/dL}$	= 1.245 $\mu\text{mol/L}$

REFERENCES: APPENDICES

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- Adriano, D.C. (1986) In: Trace Elements in the Terrestrial Environment. Springer-Verlag, New York pp. 219-262.
- Agency for Toxic Substances and Disease Registry (ATSDR), 1988. Toxicological Profile for Lead. Oak Ridge National Laboratory, Oak Ridge, TN.
- Agency for Toxic Substances and Disease Registry, 1991 (Draft). Toxicological Profile for Lead. pg 195-201.
- Agriculture Canada, 1974. Report from Plant Product Division, Production and Marketing Branch, Department of Agriculture, Ottawa, Ontario.
- Allan, L. J., 1978. Food of ring-billed and herring gulls nesting on Chantry Island, Lake Huron, 1978. CWS Wildlife Toxicology Division, Manuscript Report 39, 14pp.
- Allan, R. J., Mudroch, A. and Munawar, M., 1983. The Niagara River-Lake Ontario pollution problem. *J. Great Lakes Res.* 9(2): 109-340.
- Baumhardt, G. R. and Welch, L. F., 1972. Lead uptake and corn growth with soil-applied lead. *J. Environ. Qual.* 1(1): 92-94.
- Beyer, W. N., Chaney, R. L. and Mulhern, B. M., 1982. Heavy metal concentrations in earthworms from soil amended with sewage sludge. *J. Environ. Qual.* 11(3): 381-385.
- Bigauskas, J., 1985. Canadian Minerals Yearbook: 1985, Mineral Report No. 34. Energy, Mines and Resources Canada, Ottawa 33pp.
- Bisessar, S. and McIlveen, W. D., 1991a (draft). Trace element uptake in herbs grown on soil contaminated by a secondary lead smelter. Report to the Ontario Ministry of the Environment, in press.
- Bisessar, S. and McIlveen, W. D., 1991b (draft). Lead and other trace element concentrations in cabbage and carrot grown on soil contaminated by a secondary lead smelter. Report to the Ontario Ministry of the Environment, in press.
- Bisessar, S. and McIlveen, W. D., 1991c (draft). Uptake and toxicity of lead and other elements by lettuce and beet grown on soil contaminated by emissions from a secondary lead smelter. Report to the Ontario Ministry of the Environment, in press.

-
- Black, S. A. and Schmidtke, N. W., 1984. Overview of Canadian Sludge Handling and Lead Disposal Practices and Research. Sludge Handling and Disposal Seminar, Environment Canada - Ontario Ministry of the Environment, held at Toronto, September 18-19, 1984.
- Canada Gazette, 1989. Secondary Lead Smelter Release Interim Order. Part 1, 123(2): 33-39.
- Bondy, S. C., 1988. The Neurotoxicity of Organic and Inorganic Lead in Metal Neurotoxicity, S. C. Bondy and K.N. Presad, (eds.), CRC Press Inc, Boca Raton, pp.1-17.
- Cahill, R. A., 1981. Geochemistry of Recent Lake Michigan Sediments. Illinois Geological Survey. Technical Survey 517. 94pp
- Canadian Centre for Inland Waters, 1987a. Metal Analysis by ICP.
- Canadian Centre for Inland Waters, 1987b. Trace Metals in Fish, Atomic Absorption.
- Canadian Food and Drug Act and Regulations, 1986. pg 65A.
- Canada Gazette, Part I, 1988. Food and Drug Regulations - Amendment (Schedule No. 692). July 2, 1988. pg 2605-2607.
- Canada Gazette, Part I, 1989. Secondary Lead Smelter Release Interim Order, March 1, 1989. Extra No. 2, Vol. 123. pg 33-39.
- Canada Gazette, Part II., 1990. Department of Environment. Gasline Regulations, Part II, May 9, 1990. Vol. 1, 24, No. 10. pg 1700-1712.
- Canadian Hazardous Products Act, March 1986, 19pp.
- Cannon, H.L., 1976. Lead in vegetation. In: Lead in the Environment, Lovering, T.G., Ed. U.S. Geol. Surv. Prof Pap, 957, 53-72.
- Cantox, 1987. Potential health hazard assessment of three City of Toronto incinerator facilities. Report by CanTox Inc., Consultants in Toxicology, Oakville, Ontario, Oct. 1987 pp. B-11-1 to B-11-6.
- Centers for Disease Control (CDC), 1985. Preventing lead poisoning in young children: a statement by the Centers for Disease Control. Atlanta, Georgia.
- Centers for Disease Control (CDC), 1991. Preventing lead poisoning in young children: A statement by the Centers for Disease Control. Atlanta, Georgia. 107pp.
- Chan, C. H., 1980. St. Lawrence River Water Quality Surveys, 1977. Burlington ON: Report to the Inland Waters Directorate, Water Quality Branch. Scientific Series 113. 16pp
-

-
- Chau, Y. K., Wong, P. T. S., Bengert, G. A., Dunn, J.L. and Glen, B., 1985. Occurrence of alkyllead compounds in the Detroit and St. Clair Rivers. *J. Great Lakes Res.* 11(3): 313-319.
- Chisholm, D., 1972. Lead, arsenic, and copper content of crops grown on lead arsenate treated and untreated soils. *Can. J. Plant Sci.* 52: 583-588.
- Chisholm, D. and Bishop, R. F., 1967. Lead accumulation in Nova Scotia orchard soils. *Phytoprotection* 48: 78-81.
- City of Toronto Department of the City Clerk, Board of Health, 1988. Report 2: Environmental Health Effects of Waste Incineration in the City of Toronto, January, 1988.
- Clark, T. and Diamond, M., 1988 (draft). The feasibility of using evaluative models in the development of Canadian Sediment Quality Guidelines, 34pp.
- Clement, R. and Kagel, R., 1990. Emissions from Combustion Processes: Origin, Measurement, Control, R. Clement and R. Kagel (ed.), Lewis Publishers, Inc., Chelsea, Michigan, 1990. 491pp.
- Connors, P. G., Anderlini, V. C., Risebrough, R.W., Gilbertson, M., and Hays, H., 1975. Investigations of metals in common tern populations. *Can. Field Nat.* 89: 157-162.
- Coschi, F. Personal communication, Waste Management Branch, Ontario Ministry of the Environment.
- Cotton, R. F. and Wilkinson, G., 1988. Advanced Inorganic Chemistry, 5th edition, J. Wiley and Sons, New York, pp. 265-304.
- Custer, T. W., Franson, J. C. and Pattee, O. H., 1984. Tissue lead distribution and hematologic effects in American kestrels (*Falco sparverius*) fed biologically incorporated lead. *J. Wildl. Dis.* 20(1): 39-43.
- Czuba, M. and Hutchinson, T. C., 1980. Copper and lead levels in crops and soils of the Holland Marsh area - Ontario. *J. Environ. Qual.* 9(4): 566-574.
- Dabeka, R.W. and Mackenzie, A.D. (1987) Survey of lead, cadmium, cobalt and nickel in infant formula and evaporated milks and estimates of dietary intakes of elements by infants 0-12 months. *Sci. Total Env.* 89:279-289.
- Dabeka, R.W., McKenzie, A.D. and Lacroix, G.M.A., 1987. Dietary intake of lead, cadmium, arsenic and fluoride by Canadian adults: a 24-hour duplicate diet study. *Food Additives and Contaminants*, 4:1:89-102.
- de Treville, R.T., 1964. Natural Occurrence of Lead. *Archives of Environmental Health.* 8:24-33.
-

-
- DeLeacy, E. A., 1987. Lead crystal decanters - a health risk. *The Medical Journal of Australia* 147:622, December 7/21.
- Demayo, A., Taylor, M. C., Taylor, K. W. and Hodson, P. V., 1982. Toxic effects of lead and lead compounds on human health, aquatic life, wildlife, plants, and livestock. *CRC Critical Reviews in Environmental Control* 12(4): 257-305.
- Devault, D. S., Willford, W. A., Helleberg, R. J., Nortrup, D.A., Rundberg, E.G.S., Alwan, A.K. and Bautista, C., 1986. Contaminant trends in lake trout (*Salvelinus namaycush*) from the Upper Great Lakes. *Arch. Environ. Contam. Toxicol.* 15: 349-356.
- DiGiulio, R. T. and Scanlon, P. F., 1984. Heavy metals in tissues of waterfowl from the Chesapeake Bay. U.S.A. *Environ. Pollut. (Series A)* 35: 29-48.
- Dollard, G.J., 1986. Glasshouse Experiments on the uptake of foliar applied lead. *Environ. Pollut.*, 40:109-119.
- Drescher, H. E., Harms, U. and Huschenbeth, E., 1977. Organochlorines and heavy metals in the harbour seal, *Phoca vitulina*, from the German North Sea coast. *Mar. Biol. (Berl.)* 41: 99-106.
- Duncan, C., Kusiak, R., O'Heany, J., Smith, L., Smith, J., Spielberg, L., 1985. Blood Lead and Associated Risk Factors in Ontario Children, 1984. Ontario Ministry of Health, Ministry of Labour and Ministry of the Environment.
- Eisenreich, S.L., 1982. Atmospheric role in trace metal exchange at the air-water interface. *Jour. Great Lakes Res* 8(2):243-256.
- Eisenreich, S. J., Willford, W. A., and Strachan, M. J., 1989. The role of atmospheric deposition in organic contaminant cycling in large lakes. In: *Intermedia Pollutant Transport Modelling and Field Measurements*, D. T. Allen, Y. Cohen and I. R. Kaplan (eds.), pp. 19-40.
- Eisler, R., 1988. Lead Hazards to Fish, Wildlife, and Invertebrates: A Synoptic Review. Fish and Wildlife Services, U.S. Department of the Interior, Biological Report 85(1:14).
- Eliás, R. W., 1985. Lead exposures in the human environment. In: *Dietary and Environmental Lead: Human Health Effects*, K. R. Mahaffey (ed.), Elsevier, Amsterdam.
- Environment Canada, 1985. Tetraethyl Lead: Environmental and Technical Information for Problem Spills. Environmental Protection Service, Ottawa, 86pp.
- Environment Canada, Department of Fisheries and Oceans, and Health and Welfare Canada, 1991a. Toxic Chemicals in the Great Lakes and Associated Effects; Synopsis, Vol. I.
-

-
- Environment Canada, Department of Fisheries and Oceans, Health and Welfare Canada, 1991b. Toxic Chemicals in the Great Lakes and Associated Effects: Contaminant Levels and Trends. Vol II.
- Evans, R. D. and Rigler, F. H., 1985. Long distance transport of anthropogenic lead measured by lake sediments. *Water, Air, Soil Pollut.* 24:141-151.
- Falcone, F., 1991. Migration of lead into alcoholic beverages during storage in lead crystal decanters. *Journal of Food Protection* 54(5): 378-380.
- Fallis, unpublished data.
- Feldman, R. G., 1978. urban lead mining: lead intoxication among deleaders. *New Engl. J. Med.* 298: 1143-1145.
- Fogget, R. Personal communication. Waste Management Branch, Ontario Ministry of the Environment.
- Forbes, R. M. and Sanderson, G. C., 1978. Lead toxicity in domestic animals and wildlife. In: The Biogeochemistry of Lead in the Environment, J. O. Nriagu (ed.), Elsevier/North Holland Biomedical Press, pp. 225-277.
- Frank, R., Ishida, K. and Suda, P., 1976. Metals in agricultural soils of Ontario. *Can. J. Soil Sci.* 56: 181-196.
- Frank, R., Holdrinet, M. V., Braun, H. E. Thomas, R.L., Kemp, L.W., and Jaquet, J.M., 1977. Organochlorine insecticides and PCB's in sediments of Lake St. Clair (1970 and 1974) and Lake Erie (1971). *Sci. Total Environ.* 8: 205-227.
- Franklin Associates Ltd., 1989. Characterization of products containing lead and cadmium in municipal solid waste in the United States, 1970-2000. *Toxic Substances Journal* 9: 137-144.
- Freedman, B. and Hutchinson, T. C., 1981. Sources of metal and elemental contamination of terrestrial environments. In: Effects of Heavy Metal Pollution Plants, Vol. 2, Metals in the Environment, N. W. Lepp (ed.), Applied Science Publishers, pp. 35-94.
- Getz, L. L., Best, L. B. and Prather, M., 1977a. Lead in urban and rural song birds. *Environ. Pollut.* 12: 235-239.
- Getz, L. L., Haney, A. W., Larimore, R. W., McNurney, J.W., Leland, H.V., Price, P.W., Rolfe, G.L., Wortman, R.L., Hudson, J.L., Solomon, R.L., and Reinbold, K.A., 1977b. Transport and distribution in a watershed ecosystem. In: Lead in the Environment, W. R. Boggess (ed.), Nat. Sci. Found. Rep., NSF/RA770214, pp. 105-134.
-

Gibson, J., personal communication.

Gish, C. D. and Christensen, R. E., 1973. Cadmium, nickel, lead, and zinc in earthworms from roadside soil. *Environ. Sci. Technol.* 7(11):1060-1062.

Glass, G.E., Sorensen, J.A., and Rapp, G., 1988. Causes and mitigation of toxic contamination of the fishery in the Saint Louis River/Duluth (MN)-Superior (WI) Harbor: Fishery Nursery Area for the Western Arm of Lake Superior. 31st Conference on Great Lakes Research, May 17-20, 1988. Hamilton, ON. International Association for Great Lakes Research.

Goldberg, E. D., Hodge, V. F., Griffin, J. J. and Kaide, M., 1981. Impact of fossil fuel combustion on the sediments of Lake Michigan. *Environ. Sci. Tech.* 15(4):466-471..

Grandjean, P. and Olsen, B., 1984. Chapter 4. lead. In: Techniques and Instrumentation in Analytical Chemistry, Vol. 4. Evaluation of Analytical Methods in Biological Systems. Part B. Hazardous Metals in Human Toxicology, Elsevier Science, pp. 153 - 169.

Gray, A., 1985. Dominion Colour Company Ltd. Submission to the Royal Society of Canada Commission on Lead in the Environment, Toronto, May, 1985.

Graziano, J. H. and Blum, C., 1991a. Lead exposure from lead crystal. *The Lancet* 337: 141-142.

Graziano, J. H., Slavkovic, V. and Blum, C., 1991b. (in press) Lead Crystal: An Important Potential Source of Lead Exposure. In Press: Proceedings of the Symposium on the Bioavailability and Dietary Uptake of Lead, September, 1990. 17pp.

Harrison, R. M. and Laxen, D. P. H., 1981. Human exposure to lead and its effects. In: Lead Pollution - Causes and Control, Chapman and Hall, London. pp. 133-158.

Hilborn, J. and Still, M., 1990. A state of the Environment Report: Canadian Perspectives on Air Pollution. Environment Canada, SOE Report 90-1, September 1990. 81pp.

Hutchinson *et al.*, 1974 as cited In: The Biogeochemistry of Lead in the Environment, J. O. Nriagu (ed.), Elsevier/North Holland Biomedical Press

ICRP (International Committee on Radiological Protection) (1975) Report of the Task Group on Reference Man: Report #23. Pergamon Press, New York.

Jan, W., and Sheffield, A., 1983. National Inventory of Sources and Emissions of lead (1978). Environment Canada, Report EPS 3/EP/83/6, November, 1983. 27pp.

Hayton, A., 1988. Personal communication. Aquatic Biology Section, Water Resources Branch, Ontario Ministry of the Environment, Toronto.

-
- Health & Welfare Canada Guidelines for Canadian Drinking Water Quality, 1989. Fourth Edition.
- Health and Welfare Canada (1977) Food Consumption Patterns Report. Nutrition Canada, Ottawa.
- Health and Welfare Canada (1983) Tapwater Consumption in Canada. Report 82-EHD-80, Environmental Health Directorate, Ottawa.
- Health and Welfare Canada, HWC Draft. Reference Values for Canadian Populations
- Hemphill, D.D. and Rule, J.H., 1975. Foliar uptake and translocation of ^{210}Pb and ^{109}Cd by plants. In: Hutchinson, T.O. ed. Proceedings of the International Conference on Heavy Metals in the Environment, Toronto, October 1975. Vol III pp 77-86.
- Hesselberg, R. J., Hickey, J.P., Nortrup, D. A. and Willford, W. A., 1990. Contaminant residues in the bloater (*Coregonus hoyi*) of Lake Michigan 1969-1986. J. Great Lakes Res. pg 121-129.
- Hodson, P., Whittle, D.M., Wong, P.T.S., Borgmann, U., Thomas, R.L., Chau, Y.K., Nriagu, J.O., and Hallet, D.J., 1984. Lead contamination of the Great Lakes and its potential effects on aquatic biota. In: Toxic Contaminants in the Great Lakes, J. O. Nriagu and M. S. Simmons (eds.), John Wiley & Sons, Toronto. pp 335-369.
- Hughes, M.K., Lepp, N.W. and Phipps, D.A., 1980. Aerial heavy metal pollution and terrestrial ecosystems. *Advances in Ecological Research*, 11:217-327.
- Inventory of Great Lakes Water Quality Criteria, Guidelines, Objectives, Rules and Standards (Draft).
- Ireland, M. P., 1975. The effect of the earthworm *Dendrobaena rubida* on the solubility of lead, zinc, and calcium in heavy metal contaminated soil in Wales. *J. Soil Sci.* 26(3): 313-318.
- Ireland, M. P. and Richards, K. S., 1977. The occurrence and localization of heavy metals and glycogen in the earthworms *Lumbricus rubellus* and *Dendrobaena rubida* from a heavy metal site. *Histochem.* 51: 153-166.
- Jaques, A. P., 1985a. National Inventory of Sources and Releases of Lead (1982), Environment Canada, Report EPS 5/HA/3, September, 1985. 39pp.
- Jaques, A. P., 1985b. Summary of Emissions of Antimony, Arsenic, Cadmium, Copper, Lead, Manganese, Mercury, and Nickel in Canada. Environmental Analysis Branch, Environment Canada, Ottawa.
- Jaques, A. P., 1988. Personal communication.
-

-
- Johansen, P., Kapel, F. O. and Kraul, I. 1980. Heavy metal and organochlorines in marine mammals from Greenland. *Int. Council. Explor. Sea ICES C.M.* 1980/E:32.
- John, M. K., 1977. Varietal response to lead by lettuce. *Water, Air, Soil Pollut.* 8: 133-144.
- Johnson, A. and Cox, C., 1988. Personal communication. Aquatic Biology Section, Water Resources Branch, Ontario Ministry of the Environment, Toronto.
- Jones, J.S. and Hatch, M.B., 1945. Spray residues and crop assimilation of arsenic and lead. *Soil Sci.* 60:277-288.
- Kabata-Pendias, A. and Pendias, H. (1984). V. Lead In: *Trace Elements in Soils and Plants*. CRC Press, Boca Raton, Florida pp 154-158.
- Kay, S.H. and Haller, W.T., 1986. Heavy metal bioaccumulation and effects on waterhyacinth weevils, *Neochetina eichhorniae*, feeding on waterhyacinth, *Eichhornia crassipes*. *Bull. Environ. Contam. Toxicol.*, 37:239-245.
- Keefer, R. F., Singh, R. N. and Horvath, D. J., 1986. Chemical composition of vegetables grown on an agricultural soil amended with sewage sludges. *J. Environ. Qual.* 15(2): 146-152.
- Kemp, A. L., Williams, J. D., Thomas, R. L. and Gregory, M. L., 1978. Impact of man's activities on the chemical composition of the sediments of Lakes Superior and Huron. *Water Air Soil Poll.* 10: 381-402.
- Kemp, A. L. and Thomas, R. L., 1976. Impact of man's activities on the chemical composition in the sediments of Lakes Ontario, Erie and Huron. *Water Air Soil Poll.* 5:469-490.
- Kendall, R. J. and Scanlon, P. F., 1981. Effects of chronic lead ingestion and reproductive characteristics of ringed turtle doves (*Streptopelia risoria*) and on tissue lead concentrations of adults and their progeny. *Environ. Pollut. (Series A)* 26: 203-213.
- Kendall, R. J., Norman, G. W. and Scanlon, P. F., 1984. Lead concentrations in ruffed grouse collected from southwestern Virginia. *Northwest Sci.* 58(1):14-17.
- Kiely, P., 1991. Personal communication. Air Resources Branch, Ontario Ministry of the Environment.
- Killackey, B. Personal communication, Waste Management Branch, Ontario Ministry of the Environment.
- Korzun, E. A. and Heck, H. H. Sources and fates of lead and cadmium in municipal solid waste. *Journal of the Air and Waste Management Association* 40(9) 1220-1226.
-

-
- Koslow, E.E., Smith, W.H. and Staskawicz, B.J., 1977. Lead-containing particles on urban lead surfaces. *Environ. Sci Technol.* 11(10):1019-1021.
- Kovalevskii, A.L., 1979. Biogeochemical exploration for mineral deposits, published for the USDI and the NSF. *Amer. Ind. Publ. Co. PVT LTD.*, New Dehli. 136pp.
- Kruse, E. A. and Barrett, G. W., 1985. Effects of municipal sludge and fertilizer on heavy metal accumulation in earthworms. *Environ. Pollut. (Series A)* 38: 235-244.
- Kucera, E., 1983. Lead Distribution in Winnipeg as Reflected by City Area Dogs. Manitoba Department of Environment and Workplace Safety and Health, Environmental Management Division, Terrestrial Standards and Studies Section. Report 83-10. 25pp.
- Kuntz, K. W., 1984. Toxic Contaminants in the Niagara River, 1975-1982. Report to the Burlington, ON: Inland Waters Directorate, Water Quality Branch, No. 134, 47pp.
- Kuntz, K. W., 1988a. Contaminants in Bottom Sediments of the St. Lawrence River in June 1975. Report to the Burlington, ON: Inland Waters Directorate, Water Quality Branch, No. 147, 18pp.
- Kuntz, K. W., 1988b. Recent Trends in Water Quality of the Niagara River. Report to the Burlington, ON: Inland Waters Directorate, Water Quality Branch, No. 146, 15pp.
- Lagerwerff, J.V., 1967. Heavy-metal contamination of soils in "Agriculture and the quality of our environment" American Association for the Advancement of Science. Publication 85, pg 343-364.
- Law, S. L., and Gordon, G. E., 1979. *Sources of Metals in Municipal Incinerator Emissions* *Environmental Science & Technology* 13(4) 432-438.
- Lecos, C. W., 1987. Pretty poison: lead and ceramic ware. *FDA Consumer* July/August: 6-9.
- Linzon, S. N., Chai, B. L., Temple, P. J., Pearson, R.G. and Smith, M.L., 1976. Lead contamination of urban soils and vegetation by emissions from secondary lead industries. *J. Air Pollut. Contr. Assoc.* 26(7): 650:654.
- Liquor Control Board of Ontario (LCBO), 1991. unpublished data.
- Longcore, J. R., Locke, L. N., Bagley, G. E. and Andrews, R., 1974. Significance of Lead Residues in Mallard Tissues. *Spec. Sci. Rep.*, U.S. Fish and Wildlife Service, Wildl. No. 182, Washington, D.C. 24pp.
-

-
- Lum, K. R. and Gammon, K. L., 1985. Geochemical availability of some trace and major elements in surficial sediments of the Detroit River and western Lake Erie. *J. Great Lakes Res.* 11(3): 328-338.
- Lynch, D. W., 1973. Selected Toxic Metals in Ohio's Upland Wildlife. M.Sc. Thesis, Ohio State University, Ohio.
- MacKay, D. and Diamond, M., 1989. Application of the QWASI (Quantitative Water Air Sediment Interaction) fugacity model to the dynamics of organic and inorganic chemicals in lakes. *Chemosphere* 18(7-8): 1343-1365.
- MacPherson, A.S., 1985. Submission to the Royal Society of Canada, Commission on lead in the environment. City of Toronto Department of Public Health.
- Mahaffey, K.R. 1978. Environmental exposure to lead. In: *The Biogeochemistry of Lead in the Environment*, Vol. 1B. pp. 1-36.
- Malone, G., Koeppe, D.E., and Miller, R.J., 1974. Localization of lead accumulated by corn plants. *Plant Physiol* 53:388-394.
- Marsalek, J., 1986. Municipal sources of selected trace organics in Sarnia. *Water Poll. Res. Can.* 21(3): 422-432.
- McIver, B. and Capowski, R., 1988. Your Lead-Acid Batteries - A Negative Charge to the Environment. 10th Canadian Waste Management Conference, 1988 (*not in text*).
- McKinnon, J. G., Hoff, G. L., Bigler, W. J. and Prather, E. C., 1976. Heavy metal concentrations in kidneys of urban gray squirrels. *J. Wildl. Dis.* 12: 367-371.
- Meranger J. C., Subramanian, K.S., Langford, C.H. and Umbrasas, R. (1984) Use of an On Site Integrated Pump Sampler for Estimation of Total Daily Intake of Some Metals from Tap Water. *Intern. J. Envir. Anal. Chem* 17:307-314
- Merck Index: An Encyclopedia of Chemicals, Drugs and Biologicals, 1983. 10th edition, M. Winholz, S. Budavari, R. Blumetti, and E. Otterbein, eds., pp. 776-777.
- Mierau, G. W. and Favara, B. E., 1975. Lead poisoning in roadside populations of deer mice. *Environ. Pollut.* 8: 55-64.
- Miller, J. E., Hassett, J. J. and Koeppe, D. E., 1975. The effect of soil lead sorption capacity on the uptake of lead by corn. *Commun. Soil Sci. Plant Anal.* 6(4):349-358.
-

-
- Moore, J.F., Goyer, R.A. and Wilson, M., 1973. Lead-Induced Inclusion Bodies. Solubility, Amino Acid Content, and Relationship to Residual Acidic Nuclear Proteins. *Lab. Invest.* 29(5): 488-494.
- Motto, H. L., Daines, R. H., Chilko, D. M. and Motto, C. K., 1970. Lead in soils and plants: its relationship to traffic volume and proximity to highways. *Environ. Sci. Technol.* 4(3): 231-238.
- Mudroch, A., 1985a. Geochemistry of the Detroit River sediments. *J. Great Lakes Res.* 11(3): 193-200.
- Mudroch, A., Sarazin, L., Lomas, T., 1985b. Report on the Progress of the Revision of the MOE Guidelines for Dredged Materials Open Water Disposal, 1984-1985. Burlington, ON: Natural Water Research Institute, Contribution 85-80.
- Mudroch, A., Sarazin, L. and Lomas, J., 1988. Summary of surface and background concentrations of selected elements in the Great Lakes sediment. *J. Great Lakes Res.* 14(2): 241-251.
- Murray, C. L., 1987. The Gray Squirrel (*Sciurus carolinensis*) as a Biomonitor of Lead and Cadmium. M.Sc. Thesis, Institute for Environmental Studies, University of Toronto, Toronto, Ontario.
- National Academy of Sciences, 1980. Lead in the Human Environment. NCS Committee on Lead in the Human Environment, Washington, D.C. 517pp.
- National Incinerator Testing and Evaluation Program (NITEP), 1985: Two Stage Combustion (Prince Edward Island). Report EPS3/UP/1. September 1985. Environment Canada, Environmental Protection Service.
- National Incinerator Testing and Evaluation Program (NITEP), 1988: The Combustion Characterization of Mass Burning Incinerator Technology, Quebec City. Volume VI: Assessment of Contaminant Leachability from Residuals, August, 1988.
- National Incinerator Testing and Evaluation Program (NITEP), 1988: Environmental Characterization of Mass Burning Incineration Technology at Quebec City, Summary Report, June, 1988. 97pp.
- National Incinerator Testing and Evaluation Program (NITEP), 1989. The Combustion Characterization of Mass Burning. Incinerator Technology, Quebec City, Volume VII Evaluation of Solidified Electrostatic Precipitator Ash from a Mass Burning Municipal Waste Incinerator.
-

-
- National Incinerator Testing and Evaluation Program (NITEP), 1989: Evaluation of Contaminant Leachability from Residues Collected at a Refuse Derived Fuel Municipal Waste Combustion Facility, July 1989 (SWARU, Hamilton).
- National Incinerator Testing and Evaluation Program (NITEP), 1989: Characterization of Residues from a Modular Municipal Waste Incinerator with Lime-Based Air Pollution Control, September 1989 (Victoria Hospital, London).
- National Research Council of Canada (NRCC), 1973. Lead in the Canadian Environment. NRCC Associate Committee on Scientific Criteria for Environmental Quality, National Research Council, Ottawa. 116pp.
- National Research Council of Canada (NRCC), 1978. Effects of Lead in the Canadian Environment, Ottawa, Canada. NRCC No. 16745. 22pp.
- Neilson, M. A., 1983. Trace Metals in Lake Ontario, 1979. Burlington, ON: Inland Waters Directorate (Ontario), Water Quality Branch, No. 133. 13pp.
- Niagara River Toxics Committee (NRTC), 1984. Report of the Niagara River Toxics Committee. U. S. EPA, MOE, Environment Canada, NYDEC.
- Nixon, J., personal communication.
- Nriagu, J. O., 1979. Global inventory of natural and anthropogenic emissions of trace metals to the atmosphere. *Nature* 279: 409-411.
- Nriagu, J. O., 1985. (Editor's note: I do not have the reference for this citation from Hugh Graham's portion of the text.) ()
- Nriagu, J. O., 1986a. Global lead cycle and the Canadian contribution to it. In: Pathways, Cycling and Transformation of Lead in the Environment, P. M. Stokes, (ed.), Royal Society of Canada Commission on Lead in the Environment, Toronto, pp. 17-36.
- Nriagu, J. O., 1986b. Lead contamination of the Canadian environment. In: Health Effects of Lead, M. C. B. Hotz (ed.), Royal Society of Canada Commission on Lead in the Environment, Toronto, pp. 61-78.
- Nriagu, J. O., 1989. A global assessment of natural sources of atmospheric trace metals. *Nature* 338: 47-49.
- Nriagu, J. O. and Pacyna, J. M., 1988. Quantitative assessment of worldwide contamination of air, water and soils by trace metals. *Nature* 333: 134-139.
-

-
- Nraigu, J. O., Wong, H. K. and Coker, R. O., 1981. Particulate and dissolved trace metals in Lake Ontario. *Water Res.* 15: 91-96.
- Nutrition Canada (1977) Food Consumption Patterns report. Bureau of Nutritional sciences, Health Protection Branch, Health and Welfare Canada.
- Occupational Safety and Health Administration Air Regulations. Section 1910-1025.
- Ont. Reg. 734/88, 1988. Regulation to Amend Ontario Regulation 815/84 made under the Ontario Water Resources Act.
- Ontario Ministry of Agriculture and Food (OMAF), Ontario Ministry of the Environment (MOEE) and Ontario Ministry of Health (OMOH), 1986. Guidelines for Sewage Sludge Utilization on Agricultural Lands. Ontario Ministries of Agriculture and Food, Environment, and Health, Toronto. 32pp.
- Ontario Ministry of the Environment (MOEE), 1979a. Method for High Volume Sampling and Determination of Total Suspended Particulate Matter in Ambient Air. Report #AMP-101.
- Ontario Ministry of the Environment (MOEE), 1979b. Method for Determination of Heavy Metals in Total Suspended Particulate Matter in Ambient Air, Report #AMP-105.
- Ontario Ministry of the Environment (MOEE), 1981a. Outlines of Analytical Methods, p.95.
- Ontario Ministry of the Environment (MOEE), 1981b. Guide to eating Ontario sport fish.
- Ontario Ministry of the Environment (MOEE), 1983. Field Investigation Manual.
- Ontario Ministry of the Environment (MOEE), 1985a. A Guide to the Collection and Submission of Samples for Laboratory Analysis.
- Ontario Ministry of the Environment (MOEE), 1985b. Ontario Soil Baseline Survey Analytical Data 1980-1981. Vol. 1: Soil Baseline Program. Terrestrial Effects Program, Acidic Precipitation in Ontario Study, ARB-072-85-PHYTO, A.P.I.O.S. #002/85 (Authors: Griffith, M. A., Spires, T. and Barclay, P.) 37pp.
- Ontario Ministry of the Environment (MOEE), 1985c. Ontario Soil Baseline Survey Analytical Data 1980-1981. Vol. 2: Analytical Data for Southern Ontario. Terrestrial Effects Program, Acidic Precipitation in Ontario Study, ARB-072-85-PHYTO, A.P.I.O.S. #002/85. (Authors: Griffith, M.A., Spires, T. and Barclay, P.) 358pp.
-

-
- Ontario Ministry of the Environment (MOEE), 1985d. Ontario Soil Baseline Survey Analytical Data 1980-1981. Vol. 3: Analytical Data for Northern Ontario. Terrestrial Effects Program, Acidic Precipitation in Ontario Study, . ARB-072-85-PHYTO, A.P.I.O.S. #002/85 (Authors: Griffith, M.A., Spires, T. and Barclay, P.) 159pp.
- Ontario Ministry of the Environment (MOEE), 1986a. Annual Report - Air Quality in Ontario, 1984. Air Resources Branch (Author: Kurtz and P. Kiely).
- Ontario Ministry of the Environment (MOEE) 1986b. Effect of Waste Discharges on the Water Quality of Nipigon Bay, Lake Superior, 1983. Water Resources Branch, Great Lakes Section (Author: Kirby, M. K.) 48pp.
- Ontario Ministry of the Environment (MOEE), 1986c. Derivation and Significance of MOE "Upper Limits of Normal" Contaminant Guidelines. Technical Memorandum. Air Resources Branch, Ontario Ministry of the Environment, Toronto. 3pp.
- Ontario Ministry of the Environment (MOEE), 1986d. Contamination of vegetation and soil by lead and other elements in the vicinity of the Canada Metal Company, Eastern Avenue, Toronto - 1983, 1984, 1985. Air Resources Branch, (Author: Rinne, R.J.) ARB-064-86-Phyto. 14pp.
- Ontario Ministry of the Environment (MOEE), 1986e. Contamination of vegetation and soil by lead and other elements in the vicinity of Toronto Refiners & Smelters Limited, 28 Bathurst Street, Toronto - 1983, 1984, 1985. Air Resources Branch, (Author: Rinne, R.J.) ARB-065-86-Phyto.
- Ontario Ministry of the Environment (MOEE), 1986f. Contamination of vegetation and soil by lead and other elements in the vicinity of the Tonolli Company of Canada Ltd. and Exide Canada Inc., Dixie Road, Mississauga - 1983, 1984, 1985. Air Resources Branch, (Author: Rinne, R.J.) ARB-063-86-Phyto. 17pp.
- Ontario Ministry of the Environment (MOEE), 1987a. Determination of Trace Metals in soil by ICP-AES.
- Ontario Ministry of the Environment (MOEE), 1987b. Determination of metals in Vegetation by ICP-AES.
- Ontario Ministry of the Environment (MOEE), 1987c. Determination of Heavy Metals in Biomaterials by FAAS.
- Ontario Ministry of the Environment (MOEE), 1987d. Annual Report - Air Quality in Ontario, 1985. Air Resources Branch (Author: J. Kurtz and P. Kiely).
-

-
- Ontario Ministry of the Environment (MOEE), 1987e. Review and Recommendations on a Lead in Soil Guideline. Lead in Soil Committee, Hazardous Contaminants Branch, Ontario Ministry of the Environment, Toronto. 82pp.
- Ontario Ministry of the Environment (MOEE), 1988a. Development of an ICP/MS Based Method for the Analysis of Potable Water Samples.
- Ontario Ministry of the Environment (MOEE), 1988b. Determination of Trace Metals in Surface Waters.
- Ontario Ministry of the Environment (MOEE), 1988c. Ministry of the Environment Air Quality Information System (AQUIS). (Author: Joel Kurtz).
- Ontario Ministry of the Environment (MOEE), 1988d. Air Quality in Ontario: 1987, A Review of the Air Quality Monitoring Program. Air Resources Branch. 12pp. (appendix 96pp).
- Ontario Ministry of the Environment (MOEE), 1988e. Annual Report - Air Quality in Ontario, 1986. From J. Kurtz and P. Kiely, Air Resources Branch.
- Ontario Ministry of the Environment (MOEE), 1989. unpublished data (phytotoxicology section).
- Ontario Ministry of the Environment (MOEE), 1990a. Air Quality in Ontario: 1988, A Review of the Air Quality Monitoring Program. Air Resources Branch. 12pp (appendix 100pp.)
- Ontario Ministry of the Environment (MOEE), 1990b. Estimation of the Economic Costs and Consequences of Implementing the Proposed Revision to Regulation 308. Report prepared by Hickling Management Consulting for Policy and Planning Branch, Ontario Ministry of the Environment (Final Report and Appendices A-J).
- Ontario Ministry of the Environment (MOEE), 1990c. Ontario Regulation 309: Revised Regulations of Ontario, 1980 as amended to O. Reg. 750/88 under Environmental Protection Act, February 1990.
- Ontario Ministry of the Environment (MOEE), 1990d draft. Development of the Ontario Provincial Sediment Quality Guidelines for Arsenic, Cadmium, Chromium, Copper, Iron, Lead, Manganese, Mercury, Nickel, and Zinc. Water Resources Branch, Watershed Management Section. 44pp.
- Ontario Ministry of the Environment (MOEE), 1990e. (Draft). Scientific Criteria Document for Development of Provincial Water Quality Objectives and Guidelines: Total Inorganic Lead.
- Ontario Ministry of the Environment (MOEE), 1991a. Status Report on the Effluent Monitoring Data for the Iron and Steel Sector for the Period from November 1, 1989 to October 31, 1990. 219pp.
-

-
- Ontario Ministry of the Environment (MOEE), 1991b. The Preliminary Report on the First Six Months of Process Effluent Monitoring in the MISA Pulp and Paper Sector for the Period from January 1, 1990 to June 30, 1990). 175pp.
- Ontario Ministry of the Environment (MOEE), 1991c. The Preliminary Report on the Second Six Months of Process Effluent Monitoring in the MISA Pulp and Paper Sector for the Period from July 1, 1990 to December 31, 1990). 159pp.
- Ontario Ministry of the Environment (MOEE), 1991d. Air Quality in Ontario: 1989, A Review of the Air Quality Monitoring Program, Air Resources Branch. 32pp (appendix 105pp)
- Ontario Ministry of the Environment (MOEE), 1991e. Ministry of the Environment Draft: Volume VII In-Place-Pollutants Program Report.
- Ontario Ministry of the Environment (MOEE-ARB), 1991f draft. Ontario Ministry of the Environment "Typical Ontario Range" of Chemical Parameters in Soil, Vegetation and Snow. 39pp.
- Ontario Ministry of the Environment (MOEE), 1992a. Status Report on the Effluent Monitoring Data for the Organic Chemical Manufacturing Sector for the Period from October 1, 1989 to March 31, 1990.
- Ontario Ministry of the Environment (MOEE), 1992b. unpublished data (Municipal Industrial Strategy for Abatement: Industrial)
- Ontario Ministry of the Environment (MOEE), 1992c. (In press) Twelve Month Monitoring Data Report for the Metal Casting Sector for the Period from May 1, 1990 to April 30, 1991)
- Ontario Ministry of the Environment (MOEE), 1992d. Draft. Status Report: The Metal Mining Sector Effluent Monitoring Data for the Period of February 1, 1990 to January 31, 1991.
- Ontario Ministry of the Environment (MOEE), 1992e. unpublished data (Municipal Industrial Strategy for Abatement: Municipal)
- Ontario Ministry of the Environment (MOEE-ARB), 1992f. unpublished data
- Ontario Ministry of the Environment (MOEE). Internal review of nineteen different guidelines intended to advise on environmentally safe methods of dealing with potentially contaminated sludges, soils and excavated material (including sediment).
- Ontario Ministry of Health and Ministry of the Environment (OMOH/MOEE), 1987. Draft: Blood Lead Concentrations and Associated Risk Factors in an Appropriate Sample of Northern Ontario Children.
-

-
- Ontario Ministry of Health (MOH) and Ontario Ministry of the Environment (MOEE), 1988. Northern Ontario Blood Lead Study, 1987-1988.
- ORTECH, 1988. ORTECH International. Stack Emission Testing at SWARU For the Regional Municipality of Hamilton-Wentworth. Report by ORTECH Intl., October, 1988. 41pp.
- Ozvacic, V., 1986. Heavy Metal Emissions: Ashbridges Bay Incinerator. Memorandum from Air Resources Branch, Ministry of the Environment (MOEE), April 28, 1986. 3pp.
- Ozvacic V., Wong, G., Marson, G., Clement, R., Rokosh, D., Suter, S., Horsnell, G., Hipfner, J.C., Burns, S., Corinthios, H., Young, M., Birmingham, B., 1990. Biomedical Waste Incinerator Testing Program, Chemosphere, Vol 20, Nos 10-12, pp 1801-1808.
- Page, A.L., Gande, T.J. and Joshi, M.S., 1971. Lead quantities in plants, soil and air near some major highways in southern California. *Hilgardia* 41:1-31.
- Palmer, K.T. and Kucera, C.L., 1980. Lead contamination of Sycamore and soil from lead mining and smelting operations in eastern Missouri. *Jour. Environ. Qual.* 9(1):106-110.
- Pennak, R. W., 1978. Fresh-Water Invertebrates of the United States. Second Ed., John Wiley & Sons, Toronto.
- Persaud D., Lomas, T. D. and Hayton, A., 1987. The In-Place Pollutants Program Vol. III. Phase I Studies, Report to the Ontario Ministry of the Environment.
- Presant, E. W. and Tupper, W. 1965. Trace elements in some New Brunswick soils. *Can. J. Soil Sci.* 45: 305-310.
- Prior, M. G., 1976. Lead and mercury residues in kidney and liver of Canadian slaughter animals. *Can J. Comp. Med.* 40: 9-11.
- Province of Alberta, Clean Air Act. Clean Air (Maximum Levels) Regulation being Alberta Regulation 218/75 with amendments up to and including Alberta Regulation 40/84.
- Province of Ontario: Ambient Air Quality Criteria, Standards, Tentative Design Standards, Guidelines and Provisional Guidelines.
- Province of British Columbia, Ministry of the Environment. Pollution Control Objectives.
- Rathke, D. E. (ed.), 1984. Lake Erie Intensive Study 1978-1979: Final Report. Ohio State University, Centre for Lake Erie Area Research. Clear Tech Report. No. 284. Columbus, OH.
-

-
- Reid, N., Tang, A. J. S., Orr, D. B. and Steer, P., 1988a. The Deposition of Toxic Species in Ontario. Paper No. 88-62.4 presented at the Air Pollution Control Association (APCA) Annual Meeting, Dallas, 1988. 12pp.
- Reid, N., 1988b. Personal communication.
- Remedial Action Plan for The Niagara River (Ontario) Area of Concern (draft). Ontario Ministry of the Environment, Environment Canada, Ontario Ministry of Natural Resources, and Fisheries and Oceans Canada (MOEE/ENV/MNR/FAO), 1990, pp 67-73.
- Remedial Action Plan: Bay of Quinte. Stage 1-Environmental Setting & Problem Definition (July, 1990). Remedial Action Plan Coordinating Committee
- Remedial Action Plan. Data Report: St. Lawrence River Sediment and Biological Assessment (WRB, 1990); St. Lawrence River Environmental Investigations, Vol 1 (Feb, 1988) Vol 3 (February, 1988) Vol 4 (October, 1990)
- Remedial Action Plan: Port Hope Harbour. Stage 1-Environmental Conditions and Problem Definition (January, 1990)
- Remedial Action Plan for Hamilton Harbour: Environmental Conditions and Problem Definition (March, 1989). pp. 43-49.
- Remedial Action Plan Severn Sound: Part 1-Environmental Conditions and Problem Definitions (February, 1989). pp. 45-58.
- Remedial Action Plan Wheatley Harbour: Stage 1-Environmental Conditions and Problem Definition (April, 1990). pp. 45-55.
- Remedial Action Plan Metro Toronto: Stage 1-Environmental Conditions and Problem Definition (May, 1989) pp. 57-72.
- Rinne, R.J., 1986. Recommended Soil Clean-up Criteria for Decommissioning of Industrial Lands - Background and Rationales for Development. Draft Report, Ontario Ministry of the Environment.
- Rinne, R.J., 1988. Personal communication.
- Roberts, T. M., Gizyn, W. and Hutchinson, T. C., 1974. Lead contamination of air, soil, vegetation and people in the vicinity of secondary lead smelters. In: Trace Substances in Environmental Health - VIII, D. D. Hemphill (ed.), Proceedings of the 8th Annual Conference on Trace Substances in Environmental Health, University of Missouri-Columbia, June 11, 12, 13, 1974. pg. 155-166.
-

-
- Roberts, R. D., Johnson, M. S. and Hutton, M., 1978. Lead contamination of small mammals from abandoned metalliferous mines. *Environ. Pollut.* 15: 61-69.
- Rossman, R., 1982. Trace Metal Chemistry of the Waters of Lake Huron. University of Michigan, Great Lakes Resources Division. Publication 21. Ann Arbor, MI. 44pp.
- Rossman, R., 1983. Traces Metals in Lake Huron Waters - 1980 Intensive Surveillance. University of Michigan, Great Lakes Research Division. Report No. 97. Ann Arbor, MI. 71pp.
- Rossman, R., 1984. Trace Metal Concentrations in the Offshore Waters of Lakes Erie and Michigan. Ann Arbor, MI: University of Michigan, Great Lakes Resource Division. Report 108.
- Rossman, R. 1986. Trace Metal Concentrations in Offshore Waters and Sediments of Lake Superior. Ann Arbor, MI: University of Michigan, Great Lakes Division. Report 121. 122pp.
- Rossman, R. and Barres, J., 1988. Trace element concentrations in near-surface waters of the Great Lakes and methods of collection, storage and analysis. *J. Great Lakes Res.* 14(2): 188-204.
- Royal Society of Canada (RSC), 1986. Lead in the Canadian Environment: Science and Regulation. Final Report. Royal Society of Canada Commission on Lead in the Environment, Toronto. 374pp.
- Royal Society of Canada (RSC), 1986. Extraction and Industrial Uses of Lead, In: Lead in the Canadian Environment: Science and Regulation. September 1986, pp. 31-76.
- Salouen, L.M., 1991. Personal communication. State of California, Air Resources Board.
- Samuels and Meranger, 1984. (Editor's note: I do not have the reference for this citation from Hugh Graham's portion of the text.) ()
- Scheuhammer, A. M., 1987. The chronic toxicity of aluminum, cadmium, mercury, and lead in birds: A review. *Environ. Pollut.* 46: 263-295.
- Schmidt, J. A. and Andren, A. W., 1984. Deposition of airborne metals into the Great Lakes: an evaluation of past and present estimates. In: Toxic Contaminants in the Great Lakes, J. O. Nriagu and M. S. Simmons (eds.), Wiley Interscience, New York, pp. 81-103.
- Sileo, L. and Beyer, W. N., 1985. Heavy metals in white-tailed deer living near a zinc smelter in Pennsylvania. *J. Wildl. Dis.* 21(3):289-296.
-

Simpson, K. J., 1987. Water Quality Assessment of Ten Lake Superior Embayments, Spring 1983. Ontario Ministry of the Environment, Water Resources Branch, Great Lakes Section. 54pp.

State of Minnesota: Pollution Control Agency, 1991. Lead Abatement in Soil.

Statistics Canada and Health and Welfare Canada (1981) Canada Health Survey.

Stendell, R. C., Smith, R. I., Burnham, K. P. and Chritensen, R. E., 1979. Exposure of waterfowl to lead: a nationwide survey of residues in wing bones of seven species, 1972-73. *U.S. Fish Wildl. Serv. Spec. Sci. Rep. - Wildl.* 223: 1-12

Stendell, R. C., Artmann, J. W. and Martin, E., 1980. Lead residues in sora rails from Maryland. *J. Wildl. Manage.* 44(2): 525-527.

Stevens, R. J., 1987. A Review of Lake Ontario Water Quality with Emphasis on the 1981-82 Intensive Years. IJC Report to the Surveillance Work Group, Great Lakes Water Quality Board, Windsor, Ont.

Stokes, P. M., Hutchinson, T. C. and Krauter, K., 1973. Heavy metal tolerance in algae isolated from polluted lakes near the Subury, Ontario Smelters. *Water Pollution Research in Canada.* 8: 178-201.

Stokes, P. M., 1977. A Survey of Heavy Metals in Soils, Water and Vegetation from the Davis Tannery, Kingston, Ontario.

Stokinger, H. E., 1981. The Metals in Patty's Industrial Hygiene and Toxicology, Vol. 2A, 3rd edition. F. Clayton and G. Clayton, eds., pp. 1493-2060.

Strachan and Eisenreich, 1988. Mass Balancing of Toxic Chemicals in the Great Lakes: The Role of Atmospheric Deposition, Windsor, ON: International Joint Commission. Appendix I from the Workshop on the Estimates of Atmospheric Loadings of Toxic Chemicals to the Great Lakes Basin. 113pp.

Struger, J., Elliott, J. E. and Weseloh, D. V., 1987. Metals and essential elements in herring gulls from the Great Lakes, 1983. *J. Great Lakes Res.* 13(1): 43-55.

Sylvestre, A., Kuntz, K. W. and Warry, N. D., 1987. Water Quality at the Inlet to the St. Lawrence River, 1977 to 1983. Burlington, ON: Inland Waters/Lands Directorate, Technical Bulletin 142. 58pp.

Sypher Mueller, Life Cycle of the Lead-Acid Battery in Canada. Draft report for Environment Canada, Sypher Mueller International Inc., March 31st 1988.

-
- Temple, P. J., 1974. Lead in Hamilton vegetation and soil: 1970-1973. Air Management Branch, Ontario Ministry of the Environment, Toronto.
- Thomas, R. L., 1973. The distribution of mercury in the sediments of Lake Huron. *Can. J. Earth Sci.* 10:194-204.
- Thomas, R. L., and Mudroch, A., 1979. Small Craft Harbours Sediment Survey: Lakes Ontario, Erie and St. Clair. Burlington, ON: Canada Department of Fisheries and Oceans. 149pp.
- Tonolli, 1986. Problems in the Lead-Acid Battery Recycling Industry. Presentation by Tonolli Canada Limited to The Ontario Ministry of the Environment, Education Seminar, September 11, 1986.
- United States Environmental Protection Agency (U.S.EPA), 1977. The Short-term Effects of Lead on Domestic and Wild Animals. (Author: Botts, R. P.) EPS-600/3-77-009. 29pp.
- United States Environmental Protection Agency (U.S.EPA), 1980. Sediments in Southern Lake Huron: Elemental Composition and Accumulation Rates. (Author: Robbins, J. A.) EPA 600/3-80-080, 309pp.
- United States Environmental Protection Agency (U.S.EPA), 1986a. Air Quality Criteria for Lead, EPA/600/80-83/028F Volumes I-IV.
- U.S. Environmental Protection Agency (U.S.EPA), 1986b. Biological Monitoring Techniques for Human Exposure to Industrial Chemicals. Research Triangle Park, NC.
- United States Environmental Protection Agency (U.S. EPA), 1992. (Superfund). unpublished data (ROD Database).
- United States Food and Drug Administration (U.S.FDA), 1980. Compliance program report findings: FY 77 total diet studies - adult (7320.73). U.S. Department of Health, Education and Welfare. Available from NTIS: PB82-260639.
- United States Bureau of Mines, 1972-1984. Lead. In: Minerals Yearbook, Volume 1. Metals and Minerals. U.S. Department of the Interior, Washington, DC.
- Upper Great Lakes Connecting Channels Study (UGLCCS), 1989. Final Report, Vol. I & II.
- van der Werff, M. and Pruyt, M.J., 1982. Long-term effects of heavy metals on aquatic plants. *Chemosphere* Vol. 11 No.8:727-739.
- Wade, K. J., Flanagan, J. T., Currie, A. and Curtis, D. J., 1980. Roadside gradients of lead and zinc concentrations in surface-dwelling invertebrates. *Environ. Pollut. (Series B)* 1: 87-93.
-

-
- Wagemann, R. and Muir, D. C. G., 1984. Concentrations of Heavy Metals and Organochlorines in Marine Mammals of Northern Waters: Overview and Evaluation. Can. Tech. Rep. of Fish. Aquatic Sci. No. 1279, Department of Fisheries and Oceans, Western Region, Winnipeg, Manitoba. 46pp.
- Way, C. A. and Schroder, G. D., 1982. Accumulation of lead and cadmium in wild populations of the commensal rat *Rattus norvegicus*. *Arch. Environ. Contam. Toxicol.* 11: 407-417.
- Wedding, J.B., Carlson, J.J., Stukel and Bazzaz, F.A., 1975. Aerosol deposition on plant leaves. *Environ. Sci. Technol.* 9(2):151-153.
- Weis, I. M. and Barclay, G. F., 1984. Distribution of heavy metal and organic contaminants in plants and soils of Windsor and Essex County, Ontario. Appendix VIIIb-1.
- Weisel, C., Demak, M., Marcus, S. and Goldstein, B. D., 1991. Soft plastic bread packaging: Lead content and reuse by families. *American Journal of Public Health* 81(6): 756-758.
- World Health Organization (WHO), 1977. Environmental Health Criteria. 3. Lead. World Health Organization, Geneva, Switzerland.
- Wheeler, G. L. and Rolfe, G. L., 1979. The relationship between daily traffic volume and the distribution of lead in roadside soil and vegetation. *Environ. Pollut.* 18: 265-274.
- Whittle, D.M. and Fitzsimons, J., 1983. Influence of the Niagara River on contaminant burdens of Lake Ontario biota. *J. Great Lakes Res.* 9(2):295-302.
- Wolnik, K. A., Fricke, F. L., Capar, S. G., Braude, G.L., Meyer, M.W., Satzger, R.D., and Bonnin, E., 1983. Elements in major raw agricultural crops in the United States. 1. Cadmium and lead in lettuce, peanuts, potatoes, soybeans, sweet corn, and wheat. *J. Agric. Food Chem.* 31: 1240-1244.
- Wolnik, K. A., Fricke, F. L., Capar, S. G., Meyer, M.W., Satzger, R.D., Bonnin, E., and Gaston, C.M., 1985. Elements in major raw agricultural crops in the United States. 3. Cadmium, lead, and eleven other elements in carrots, field corn, onions, rice, spinach, and tomatoes. *J. Agric. Food Chem.* 33: 807-811.
- Wong, P., 1992. personal communication. Ontario Ministry of the Environment (MOEE), Air Resources Branch.
- Wong, P. T. S., Chau, Y. K., Yaromich, J., Hodson, P. and Whittle, M., 1988. Alkyllead contaminations in the St. Lawrence River and St. Clair River (1981-1987). Department of Fisheries and Oceans, Bayfield Institute (GLLFAS), Burlington, Ontario. Canadian Technical Report of Fisheries and Aquatic Sciences, No. 1602. 134pp.
-

-
- Zakshek, E. M., Puckett, K. J. and Percy, K. E., 1986. Lichen sulphur and lead levels in relation to deposition patterns in eastern Canada. *Water, Air, Soil Pollut.* 30:161-169.
- Zimdahl, R.L., 1975. Entry and movement in vegetation of lead derived from air and soil sources. Paper Presented at the 68th Annual Meeting of the Air Pollution Control Association, Boston, Mass., June 15, 1975.
- Zimdahl, R.L. and Foster, J.M., 1976. The influence of applied phosphorus manure or lime on uptake of lead from soil. *Journal of Environmental Quality* 5(1):31-34
- Zimdahl, R.L. and Koeppe, D.E., 1977. Uptake by plants, In: Lead in the Environment, Boggess, W.R. and Wixson, B.G., Eds., Report NSF, National Science Foundation, Washington, D.C., 1977, 99-104.
- Zimdahl, R.L., and Skogerboe, R.K., 1977. Behaviour of lead in soil. *Environmental Science and Technology* Volume 11, Number 13 pp 1202-1206.
- Zimdahl, R.L. McCreary, D.T., and Gwynn, S.M., 1978. Lead uptake by plants - the influence of lead source. *Bull. Environ. Contam. Toxicol.*, 19:431-435.

